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Volume 53 JANUARY 1960 Number 1

the Journal

OF THE OKLAHOMA STATE MEDICAL ASSOCIATION



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CONTENTS

EDITORIALS

Peniwisle 1
President's Letter 36

SCIENTIFIC ARTICLES

Selected Problems of Treatment in Congestive Heart Failure, *William Finis Ewing, Jr., M.D.* 3
The Treatment of Cutaneous Pyodermas, *Mark Allen Everett, M.D.* 7

MEDICAL CENTER

Message from the Dean 9
Cardiac Arrest, *G. Rainey Williams, M.D.* 10
Ewing's Sarcoma of the Tarsal Bones—Two Case Reports, *Marvin K. Margo, M.D., and James N. Owens, Jr., M.D.* 14
Abstracts 18
New Faculty Appointments 19

SPECIAL ARTICLES

Profession or Business? *J. H. Means, M.D.* 20
Vicarious Wish Fulfillment For Young Boys and Old Boys, *Joseph J. Quinn* 29
Trends in the Distribution and Organization of Physicians in the Practice of Medicine, *L. W. Martin, Ph.D.* 31

NEWS

Actions of AMA Clinical Meeting 38
Family Doctors Schedule Philadelphia Meeting 41
Martin Comes Home 42
Detailmen Honor Martin 43
AAGP Meeting Set For February 43
AMA, AHA Pledge Health Help for the Needy 44
School Health Plan Outlined by Pediatricians 45
Tulsa County Society Welcomes New Members 47
Health Problems Discussed on New Radio Series . . . 50
Texas Announces Medical Cruise 51
Requests for *Journals* 51
Have You Heard? 51
Scientific Exhibit Application 53
Auxiliary News 54
Hobby Show Application 55
Army Needs Doctors 55
College of Surgeons to Hold Colorado Meeting 55
Coming Meetings 56
Deaths 57
25 Years Ago 58
Physician Placement 59
Miscellaneous Advertisements 60, Index to Advertisers lxii

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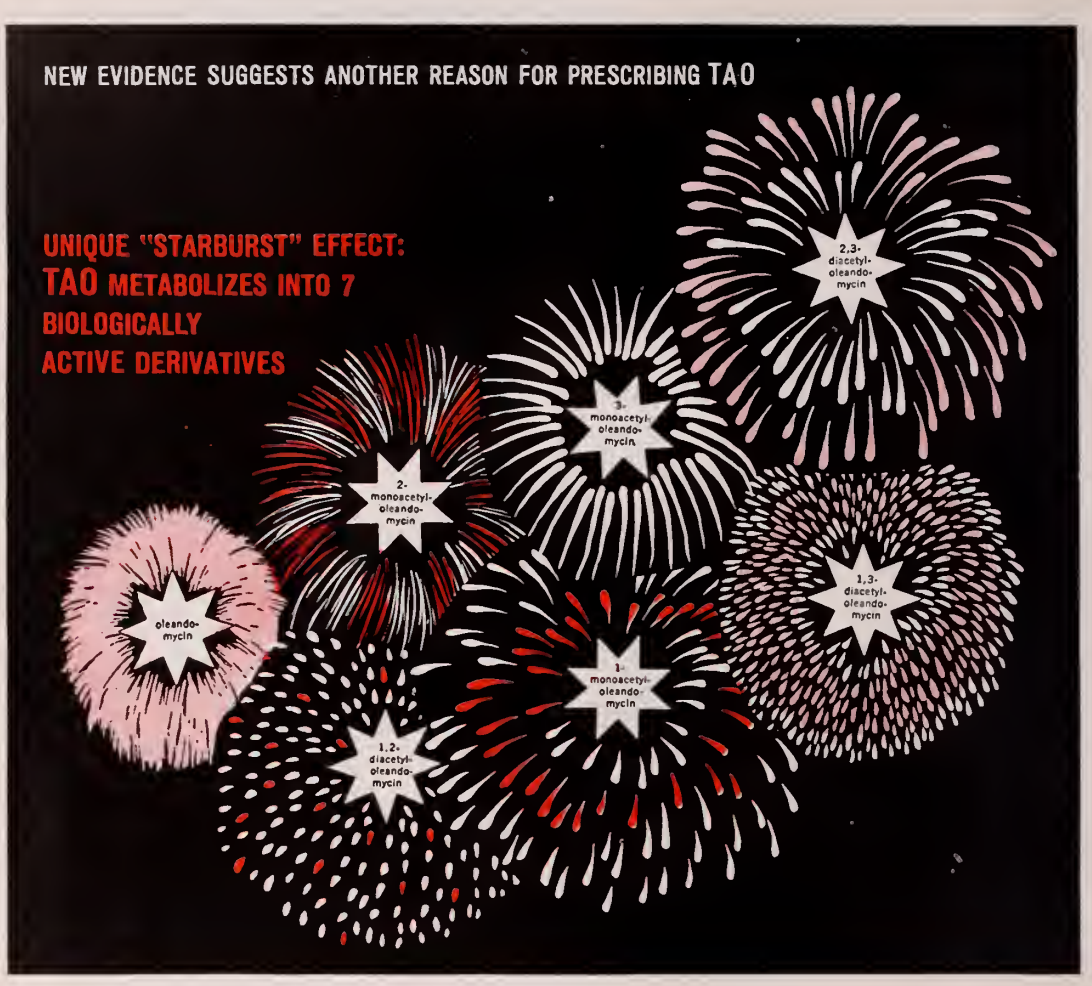
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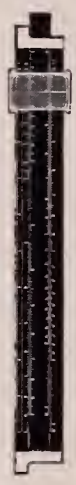
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Editorial

Peniwisle

From the fiercely glowing forges of the pharmaceutical industry has issued another significant contribution to the pressing needs of therapeutics.

This newest arrival on the medical scene is a combination of two well established therapeutic agents which, by their happy union, have provided a new "vistaspectrum" agent. It is further heartening to find two competitive drug firms combining their resources in the formulation of a product for the common good. These two products penipetit (Penisyn,[R] LaBelle Laboratories) and corticowhistle (Whi-cort,[R] J. P. Wort and Co.) are, of course, familiar items of the armamentarium.

Perhaps the first recognition of therapeutic properties of Penisyn should be credited to an otherwise unknown and untutored sheepherder in the mountainous Basque province of Northern Spain. This remarkably perceptive individual found that leaves of a plant indigenous to very high mountain slopes in his native province, when collected during the rutting season of the chamois, effectively improved the taste of his otherwise harsh wine. It was this lonely and often depressed guardian of the sheep who noted the remarkable changes in mood following the ingestion of large quantities of this crude extract. This important therapeutic principle would undoubtedly have been lost to medicine had it not been for the world wide activities of the American pharmaceutical industry. In 1942 agents of LaBelle Laboratories were diligently searching the world for the very finest sherry wines for use as a vehicle in which to make more palatable their vitamin products to a highly competitive and taste-concious market. One of these representatives happened across this very happy sheepherder in his most remote and desolate country and, according to the customs of the region was offered a cup of wine. He was immediately impressed by the pleasant taste that the exotic leaf imparted to the otherwise indifferent wine. As neither individual recognized the scientific potential of the leaf, the secret and an exclusive right to export of the product was purchased for the sum of one penny (and much labor). The plant was named Pennywort in deference to the purchase price and in honor of Mr. J. P. Wort, president of LaBelle.

Remarkable properties of the pennywort-wine-vitamin elixir sold under the trade name Winovit[R] became rapidly established. Another drug house, J. P. Wort and Co., was licensed to produce the combination under the trade name Vinvit[R] and eventually allowed marketing of a less potent over-the-counter product, Vitavin,[R] by a subsidiary company, Wort Brothers. These products have reached exceptionally wide use—particularly in the treatment of temperate older individuals.

Due to the limited supply of suitable pennywort leaves a great deal of effort was expended by scientists of LaBelle to extract, characterize and synthesize the active principle of pennywort leaves. This substantial achievement was accomplished over a period of about two years and the synthetic material assigned the generic name "penipetit" and the trade name "Penisyn.[R]" A wide variety of pharmaceutical forms of Penisyn were then made available including scored compressed tablets in strength of 10 mg., 25 mg. and 100 mg., delayed action tablets containing 200 mg. and 400 mg. of the active principle, pediatric and geriatric elixirs, a syrup suitable for use as a topping for ice cream, pessary shaped suppositories, a tincture for external use as well as other dosage forms.

The drug has become established as a wide spectrum psychotherapeutic agent admirably suitable for the treatment of the greater proportion of office patients. Moreover the drug exhibits anti-rheumatic effects particularly when administered with aspirin. It is effective as a diuretic when given with

copious quantities of water, as a harmless preanesthetic agent, as a weight reducer when combined with a 500 calorie diet and, strangely, a weight stimulator when administered with a 3500 calorie diet. It serves as an effective surface antiseptic when incorporated into 70% alcohol. It's soporific qualities are particularly evident when taken in a glass of warm milk immediately upon retiring. The remarkable synergistic and antagonistic properties of Penisyn are further evident with a wide variety of other medications. An interesting development occurred soon after introduction of the synthetic drug much to the initial concern of the company. It was discovered that Penisyn was being widely used as a substitute for Penicillin, undoubtedly due to the unfortunate similarity in names. On investigating the results of such usage the surprising fact became apparent that the drug was indeed effective in at least 60% of the cases in which penicillin would have been used.

Despite the unparalleled wide use of Penisyn virtually no toxicity has become apparent. However, reports have recently appeared indicating that the drug might have addicting properties in addiction-prone individuals after prolonged use.

As might be expected, even in this era of miracle drugs, Penisyn does have several short comings. While it frequently produces temporary beneficial effects in acute leukemia, disseminated lupus erythematosus, blastomycosis, cirrhosis of the liver, Addison's disease and other severely life-threatening diseases it has not proven curative of these diseases. Thus in a very happy exchange with J. P. Wort and Co. (which illustrates the remarkable cooperation between, at least certain, pharmaceutical houses) another agent was acquired to form the exciting combination which has been recently introduced.

The latter drug is a steroid of the glucocorticoid series known by the generic name of corticowhistle (Whi-Cort,® J. P. Wort & Co.). This agent has never reached the popular use which it deserves due to the prior introduction of a vast number of other glucocorticoids already thoroughly familiar to the physician. However it is an extremely potent anti-inflammatory agent with very little diabetogenic effect in individuals without a predisposition to diabetes, producing very little evidence of Cushing's syndrome when given for periods of less than one week and a very low incidence of symptoms characteristic of peptic ulcer.

Penipetit and corticowhistle when mixed in the dry form proved to be noninflammable, nonexplosive, nonhygroscopic, did not change color on exposure to the elements and produced no objectionable after-taste. Thus it was put on clinical trial and after exhaustive study by 10,000 physicians (of whom only 500 had the courtesy to report their results) in a wide variety of disorders was cautiously introduced on the market under the trade name of Peniwisle.®

Extensive advertising space is being immediately devoted to acquainting the medical profession with this product, largely devoted to the purpose that prescribers of the drug quickly learn to spell the rather difficult name. A large part of these full page presentations, attractively printed in red on a gold background, has been devoted to listing the names of the 500 physicians who pioneered the original clinical trials of the product. Therapeutic claims have not been extravagant and the theme has been simply stated "When in doubt—use Peniwisle.®" A simple line drawing of the serene shepherd happily fluting to his contented sheep is prominently displayed in the upper left quarter of the advertisement. A little gimmick that has been used to introduce the physician to the product is a silver flute, which can also be used as a monaural stethoscope, with the word "Peniwisle" engraved in small letters along the embouchure.—*R.W.P.*

Scientific Articles

The current status of valvular surgery and radioactive iodine therapy.

Selected Problems of Treatment in Congestive Heart Failure

WILLIAM FINIS EWING, JR., M.D.

William Finis Ewing, Jr., M.D., graduated from the University of Oklahoma School of Medicine in 1953. Doctor Ewing is now practicing in Tulsa, Oklahoma where his practice is limited to his specialty, internal medicine.

This is the fifth of a series of articles prepared for *The Journal* under the auspices of the Oklahoma Heart Association and its committee on Professional Education, emphasizing the theme of the Association for the current year. Previous related articles have touched on more conventional medical treatment of congestive failure. Doctor Ewing has selected three of the more controversial means of therapy and attempted to survey their current status. Treatment of congenital heart disease has been ignored.

Surgery in Mitral Stenosis

MITRAL VALVULAR LESIONS are the most common ones following rheumatic heart disease and of all the valvular lesions mitral stenosis has been most successfully attacked surgically so far. One of the most difficult aspects of evaluating the effect of surgical treatment of stenosis of the mitral valve is lack of knowledge of and wide variation in the course of this disease when medical management alone is used. The average age of death in people with mitral stenosis is about forty-five; 25% survive beyond fifty. A patient with the findings of mitral stenosis in his late twenties or early thirties poses a difficult prognostic problem, indeed. Certain milestones in the course of the disease portend an unfavorable prognosis; among these are atrial fibrillation, episodes of pulmonary edema, and finally right heart failure with edema. According to Wilson & Greenwood with the onset of right heart failure, 25% of the patients will be dead within six months.

Before quoting Ellis, Harken & Black in the grim matter of mortality statistics, some explanation of their classification of mitral stenosis is necessary. That classification adheres fairly closely to the New York Heart Association classification.

Class I—No symptoms.

Class II—Slight restriction in exercise tolerance because of pulmonary symptoms but no progression.

Class III—Progressive pulmonary symptoms and ordinary activity curtailed.

Class IV—Right heart failure—cardiac invalidism.

Ellis, Harkins & Black, in a review of 1,000 cases followed from one to nine years following mitral commissurotomy, had an over-all mortality of 8%. Patients in Class II and

III who made up 400 of the first 500 cases had an operative mortality of 6%. In their second 500 cases of Group II and III (375) the operative mortality was 0.8%. In Group IV (145), of the first 500 cases the mortality was 27%. In the last 500 cases reviewed of which 125 were of Group IV, the operative mortality was 19%.

Complications and morbidity related, or possibly related, to surgery consisted of a 30% incidence of the so-called "post commissurotomy syndrome." The incidence of peripheral embolization was very high in early cases; however, in the last 500 cases the over-all incidence was 3.5% with Group IV patients having much the higher rate.

If we now turn to the results of the operative procedure on the same 1,000 patients: Of Group IV patients, 100 have been followed five years and 60% are alive. This is 60% of the total number operated on and considering that 19% died at the time of operation this seems a definite improvement in longevity. Of the Group II and III patients, 400 patients have been followed five years with survival of 84% at that time (operative mortality of 6%).

Ellis and co-workers considered the patients significantly improved if in their opinion they had moved up I or II Grades in the American Heart Association Classification for heart disease. In Group II and III patients, (New York Heart Association) 83% were considered significantly improved (83% of 700 cases) at the end of one year. At the end of five years, 76% (of 400 cases) were considered improved. Again, in Group II and III at the end of eight years (29 cases) 55% were considered improved. In Group IV, after the first year, 68% (205 cases) were considered significantly improved. In both classifications, 3% were made worse. These were primarily patients in which mitral insufficiency was either made worse or induced.

Younger patients and patients with normal sinus rhythm had significantly better results than their elders and those with atrial fibrillation.

In consideration of the effect of valvulotomy, including appendage amputation and atrial flushing on embolism, let us look briefly at these figures from the same 1,000 patients: 18% had had emboli prior to operation. In the total follow-up, 2.5% have had emboli since operation at an average time of four years postoperatively (+3.5% operative emboli).

It would appear, from the above figures and considering the enthusiasm of the authors, that mitral valvulotomy is of benefit in the patient with mitral stenosis, causing decrease in symptomatology and probably prolonging life. The operative mortality of 5-6% in Group II and III patients compared to 20% in patients of Group IV indicates that patients should be operated on before they reach Group IV.

Recurrent embolism is probably an indication for valvulotomy.

In consideration of patients for operation the ideal candidate is a young person (under fifty) having progressive symptoms; having a mitral orifice of less than one square centimeter with not too much calcium in the leaflet; having no other valvular lesion and no extensive myocardial damage. Most patients do not require cardiac catheterization. A systolic murmur at the apex without any other signs of mitral insufficiency does not constitute a contraindication. Patients with associated aortic valvular disease need cardiac catheterization before a decision can be made.

Electrocardiographic evidence of LVH is a contraindication to operation for mitral stenosis. Advanced age, visible calcification, or an enlarged heart are relative contraindications.

Watt, Bigelow & Greenwood report a series of twenty-four patients who underwent mitral valvulotomy during pregnancy without mortality. They feel that patients who develop failure during pregnancy, especially episodes of pulmonary edema, should have mitral valvulotomy and that therapeutic abortion should be reserved for those who refuse mitral valvulotomy.

Radioactive Iodine Therapy

Reducing metabolic requirements to within the capabilities of the heart in an attempt

to ameliorate symptoms of congestive heart failure (and angina) is not a new idea. The obvious approach is ablation of the thyroid. In 1931 Blumgart first advocated total thyroidectomy to relieve symptoms of congestive heart failure. About 50% of his patients, he felt, got definite improvement from this method of therapy; however, operative mortality was high and the treatment was subsequently abandoned. Later, when the thiouracil group of drugs became available, these were tried. The side reactions to the drugs themselves, such as fever, rash, and the temporary nature of thyroid suppression, were drawbacks to this method of therapy. When radioactive iodine became available, Blumgart in 1948 pioneered in ablation of the thyroid with this agent. In 1955 Blumgart summarized the results of 1,000 cases treated with radioactive iodine in his and other clinics. He found that 62% of the patients with congestive heart failure had worthwhile results, and that 76% of the patients with intractable angina had worthwhile results.

In another series reported by Jaffee, of 231 patients, he found that 81% of the patients with congestive heart failure had excellent or good results. He reported an even higher figure (91%) of patients with angina who had excellent or good results. The results for patients with both congestive heart failure and angina were not appreciably different in the two series from those for congestive heart failure. Jaffee reported that 45% of his patients were dead within a period of three to four years. The exact methods of ablating the thyroid with radioactive iodine differ; however, most are similar. Initially, fairly large doses (up to 100 millicuries of radioactive iodine) were used; however, these were abandoned because of the danger of postradiation thyroiditis and a consequent transient hyperthyroid state. At the present time most clinics give radioactive iodine in doses of 6 to 10 millicuries every week or two until a total of 30 or 40 millicuries has been given. The B.M.R. and the clinical state is followed and usually at the time the patient becomes clinically hypothyroid, improvement in symptomatology, if any, is noted. If this course of radiation fails to establish hypothyroidism, a second and even a third course is given. Thereafter, patients are given thyroid in an attempt to keep the B.M.R. at about -20 or -25, in sufficient quantity to avoid the symptoms of profound hypothyroidism, while keeping the cardiac symptoms in abeyance.

Most of the patients that one would consider for this type of therapy would be those with arteriosclerotic heart disease. Patients must be fairly stable in their congestive failure picture as it takes two to four months to achieve hypothyroidism. No one claims any increase in longevity for this treatment. It is solely a symptomatic treatment. The serum cholesterol rises as myxedema is induced.

While the results obtained with this method of treatment are not uniformly good, it is not traumatic; is almost without risk to the patient and is not terribly expensive. The induced disease is easily controlled with thyroid and is worthy of consideration in patients who are fairly stable, in severe congestive failure, or those who have had severe angina for a year and are not responding to more conventional therapy.

Surgical Treatment of Aortic Stenosis

The surgical approach to acquired aortic stenosis has been disappointing. The basis of the difficulty in surgical correction is the nature of the pathologic involvement of the valve. In aortic stenosis of clearly rheumatic etiology the valve cusps are fused most heavily near the aortic wall; the entire cusps become thickened; there is usually some associated aortic insufficiency and often an associated mitral lesion; and calcification is common. In the clinically different calcific aortic stenosis, whatever its etiology, probably no valve ever becomes "tightly" stenosed without heavy calcification and often the cusps are thickened and effectively immobolized at the base. Attempting to return these valves to a functioning state has been a formidable challenge and no one feels satisfied with surgical results, thus far.

Patients with predominate aortic stenosis live longer, on the average, than patients with mitral stenosis; those with calcific aortic stenosis often live an almost normal life span. Moreover, they are apt to be symp-

tom-free for many, many years. Once patients become symptomatic with dyspnea, angina or syncope their course is uniformly and rapidly downhill. Contratto & Levine noted the occurrence of death in aortic stenosis on an average of 23 months after the onset of dyspnea and nine months after the onset of syncope. An additional problem in those of the calcific group is the question of concomitant coronary atherosclerosis and its role in symptom production.

The clinical diagnosis of aortic stenosis in the calcific group may be less accurate than a similar diagnosis of mitral stenosis. Ellis performed left heart catheterization on 60 patients with clinical "tight" aortic stenosis, measuring the gradient between left ventricle and brachial artery and blood flow by means of dye curves. He found that his clinical diagnosis coincided with the calculated valve area in 75% of the cases. The amount of aortic insufficiency is almost impossible to assess with any technique. The clinical sign most consistently denoting a small valve area was a low pulse pressure.

Abelmann, et al., report on a group of 86 patients, followed one to four years, who were operated on by Bailey with the transaortic approach. There was an operative mortailty of 17.5%. Of 71 survivors, 11 died during the first year. Of the 60 still alive, the average follow-up was 29 months and of these, 74% were better symptomatically. The figures looked at in another way —56% of those who survived operation improved. Patients in right heart failure did poorly, there being a 38% mortality and only a 25% improvement.

In the Harken series of 100 patients with calcific aortic stenosis operated on by the transaortic route, there was an operative mortality of 16%. There were 12 additional deaths within the first year after operation. Of those surviving, 25% were markedly improved; 18% were slightly or moderately improved. Of the 50 patients who had been followed more than six months, there was 86% improvement. In the last 60 patients operated on, there have been only five deaths, a mortality percentage of eight. Harken further reports 19 patients who had both mitral valvulotomy and aortic commissurotomy with a 10.5% operative mortality.

These high figures of surgical mortality are indeed formidable; however, the patients had a malignantly progressive disease. Harken points out that, of the 54 patients advised to have aortic commissurotomies who refused, 49 were dead within six months.

Given a patient with aortic stenosis, who is developing symptoms, one is faced with this problem: one can be 75% sure that symptoms are caused by a "tight" valve; if surgical intervention is carried out the patient has a 5/6 chance of surviving the operation and if he survives the operation he has approximately a 50% chance of improvement.

The first problem, that of accuracy of diagnosis, can be improved by left heart catheterization; but even then the aortic insufficiency is difficult to assess.

At this time, surgical correction of acquired aortic stenosis is to be rarely recommended.

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2020 S. Xanthus, Tulsa, Oklahoma

The Treatment of Cutaneous Pyodermas

MARK ALLEN EVERETT, M.D.*

In 1951, Mark Allen Everett, M.D., graduated from the University of Oklahoma School of Medicine where he is now an Instructor in Dermatology.

Certified by the American Board of Dermatology, Doctor Everett is a member of the American Academy of Dermatology and is now serving as President of the Oklahoma State Dermatological Society. In addition, he is Consultant in Dermatology to the United States Air Force.

ANTIBIOTICS in greasy bases, either with or without the addition of cortisone derivatives, continue to be advocated for the treatment of pyodermas. Because it is the opinion of the author that an antibiotic incorporated in a non-greasy vanishing cream is the choice topical agent in the treatment of cutaneous pyodermas, the following series of cases is presented.

Methods

Seventy patients were studied. Thirty-nine received oral antibiotics. Thirty-one received only topical therapy. Three topical agents were used: Neomycin sulfate 5 mg. per gram in a special vanishing cream base:** 0.5% aqueous gentian violet; Neomycin sulfate 5 mg. per gram and Zinc bacitracin 400 units per gram in a greasy base. All cases as well as the results are summarized in Table I. All patients received instructions to bathe daily with a hexachlorophene containing soap being careful to remove all

*University of Oklahoma School of Medicine, Department of Dermatology, John H. Lamb, M.D., Chairman.

**Prepared and furnished by Dome Chemical Company, New York City.

TABLE I

Summary of Treatment of Pyodermas

DIAGNOSIS	Total Number	Neomycin cream	Neomycin Cream plus Antibiotic	Gentian Violet plus Antibiotic	Neomycin grease	Neomycin Grease plus Antibiotic	Antibiotic Alone
Impetigo contagiosa extensive	20	1	12	3	0	3	1
Impetigo contagiosa moderate	13	9	0	0	2	0	2
Infectious eczematoid dermatitis	6	0	4	1	0	0	1
Infected eczematoid disease of hands	11	2	1	1	3	2	2
Infected eczematoid disease of legs	3	0	1	1	1	0	0
Infected eczematoid disease in atopics	5	0	1	2	0	1	1
Infected prurigo in Indians	12	4	5	0	2	1	0
Totals	70	16	24	8	8	7	7
Time in days for complete recovery	—	5-12	3-7	4-7	8-17	8-14	6-10

crusts gently. Bathing was preceded by one hour period of soaking. Drying was accomplished by patting or fanning or both. The topical medication was then applied. All patients received an antihistamine four times daily. The antibiotic therapy utilized is summarized in the table.

Results

Of patients treated with both topical and systemic antibiotics those receiving the water miscible cream or aqueous topical agents improved more rapidly and cleared more quickly than those applying topical agents in a greasy base or those receiving no topical antibiotic. Of the milder cases treated by topical applications alone those receiving the water miscible cream or aqueous product again improved more quickly. Those patients receiving oral but not topical antibiotics responded more quickly than those receiving Neomycin in grease plus antibiotics. It was noted that in those patients treated with a greasy base small pustules and vesicles continued to form under the medication for several days. This phenomenon was not observed in the cream or aqueous group. Apparent sensitivity to gentian violet was observed in one patient. No sensitization to Neomycin was observed, confirming the low index of sensitivity reported by others.[2,3]

Comments

This study re-emphasized the superiority of water miscible and aqueous products over greases in the treatment of oozing lesions of various impetigenized diseases of the skin. Progression of the process was halted sooner and clearing was more rapid. From the view point of patient acceptability there was a marked preference for the Neomycin cream inasmuch as it was odorless, non-staining, colourless, and non-greasy. Aqueous gentian violet, while effective, met with considerable patient resistance because of the staining qualities. Contact sensitivity was not a factor in short term therapy such as carried out in this study.

Summary

Patients with various impetigenous dermatoses were treated with a topical cream containing Neomycin sulfate. Clearing of the disease process was rapid and the agent was characterized by greater patient acceptability than other agents tried.

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411 N.W. 11th, Oklahoma City, Oklahoma

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MEDICAL CENTER

Cadaver Supply and the Anatomical Law

"You who investigate this our machine, let it not sadden you that you impart knowledge of it at the expense of another man's death. But rejoice that our creator has equipped the intellect to such an excellence of discernment."

These wise words of the great artist-anatomist Leonardo Da Vinci still have meaning. Even today those who are responsible for procuring human dissection material often encounter superstitions and prejudice.

The first anatomical law of the State of Oklahoma was passed by the 15th Legislature April 16, 1935. In essence, it requires that all unclaimed bodies which otherwise would be buried at public expense be turned over to the State Anatomical Board to be used for the advancement of "Medical and Anatomical Studies."

In the past this law has operated quite satisfactorily in supplying the necessary study material. But with the rise in over-all prosperity, the propagation of burial insurance and of social welfare measures providing funds for burial, and the prevalence of autopsies on mental patients and unidentified dead, a shortage of cadavers could interfere with an expanding teaching program.

The problem is, of course, nation-wide. There are already at least eight medical schools in the United States where it is no longer possible to keep up a ratio of four students per cadaver, and one school where even a ratio of eight to one cannot be maintained.

Aked in a questionnaire whether they considered their anatomical material supply was ample, representatives of almost one-third of all medical schools answered in the negative.

In Oklahoma, close liaison with superintendents of state hospitals and with responsible morticians so far has allowed us to meet our teaching requirements. The Oklahoma State Legislature has done its part to forestall an anticipated shortage. A law was passed in 1957 which can greatly alleviate the problem.

The 1957 law provides that persons who wish to donate their bodies for teaching purposes and research or parts of their bodies for therapeutic reasons can do so by simply willing them to the University of Oklahoma School of Medicine or to institutions of their choice. The law made it possible for Oklahoma Lions Clubs two years ago to establish the statewide Eye Bank at the Medical Center with the gratifying result that already more than 120 patients have been able to undergo corneal transplant operations. It also provides a source of material for bone and artery and other tissue banks that permit surgical advances in the area of tissue transplantation.

Donors have only to sign a document which is written in understandable, non-legal terms, in the presence of two witnesses. Physicians in the State of Oklahoma, who are of course best qualified to evaluate the importance of sufficient dissection material, can greatly assist in securing cadavers by informing patients of this means of contributing to the progress of medical science.

Mark R Everett

This review on the subject Cardiac Arrest was prepared for presentation at a postgraduate session of the American College of Surgeons meeting at Atlantic City, October 1959.

CARDIAC ARREST

G. RAINEY WILLIAMS, M.D.

G. Rainey Williams, M.D., graduated from Northwestern University School of Medicine in 1950. He is certified by the American Board of Surgery and the American Board of Thoracic Surgery.

Doctor Williams is Assistant Professor of Surgery at the University of Oklahoma Medical Center.

FEW TOPICS merit more frequent review than cardiac arrest. The occurrence of sudden cessation of circulation in a patient in whom it is not expected is a catastrophe which demands prompt and proper resuscitative efforts to prevent death or serious neurological injury. This catastrophe is increasing in frequency, and probably occurs between 8 and 9,000 times each year in the United States.

Investigation into the cause of circulatory arrest—and this includes ventricular fibrillation as well as cardiac standstill—reveals that, with the exception of ventricular fibrillation following electric shock, every instance of cardiac arrest appears to be due to a combination of circumstances. Some of these causes are understood and many are not. Among the agents recognized as contributing to cardiac arrest should be listed: hypoxia, hypercapnia, acidosis, vagal reflexes, and depressant drugs.

Anoxia or hypoxia has been considered by many surgeons as the most important cause of cardiac arrest. Hypoxia does not consistently result in sudden circulatory arrest in the laboratory animal, and, fortunately, cardiac arrest usually does not follow its clinical appearance. Although the association of hypoxia and circulatory arrest has been emphasized by Gerbode,[7] Johnson, and Kirby[8] its exact role as an etiologic agent is not clear. Emphasis on the prevention, recognition, and correction of hypoxia is certainly justified.

Hypercapnia, the accumulation of carbon dioxide in the body, may occur even in the presence of adequate oxygenation. Hypercapnia results in respiratory acidosis and hyperkalemia, and in its presence vagal inhibition of the heart is potentiated.[22] There is little doubt that this may contribute to circulatory arrest in some cases. It follows that hypercapnia, and especially sudden changes in CO_2 level, should be avoided.

From the Department of Surgery, University of Oklahoma Medical Center, Oklahoma City, Oklahoma.

The importance of a vagal or vago-vagal reflex in causing cardiac arrest is often discussed but remains unsettled. Stimulation of the central end of the vagus nerve will cause ventricular standstill in the experimental animal, but even with continued stimulation vagal escape occurs. There are apparently no vagal fibers of the ventricles of the human heart. Reid, Stephenson, and others conclude the vagal stimulation will not cause cardiac arrest in the normal healthy heart.[14,16] In the presence of myocardial disease or temporary myocardial depression by drugs, vagal inhibition of the heart may be of great importance.

Admitting that knowledge of the etiology of cardiac arrest is incomplete, it becomes important to recognize those situations in which it is likely to occur. The majority of instances of circulatory arrest are encountered in the operating room and at the present time occur once in approximately every 1,500 operative procedures.[4,7,17,18] It does not seem coincidental that the first recognized case of cardiac arrest was reported within a few months of the introduction of anesthesia.[2] Anesthetic procedures offer the hazard of vagal stimulation during intubation and extubation, the possibility of hypoxia and hypercapnia, and the depressant effect of anesthetic agents themselves. Beecher has pointed out the apparent increase in incidence of cardiac arrest when using multiple anesthetic agents, particularly including curare like drugs.[3]

Circulatory arrest during operation is more likely to occur when no pre-anesthetic medication is given, in overly apprehensive patients, in children, and in patients with pre-existing heart disease. Furthermore, arrest has been reported most frequently during biliary and pulmonary operations.[16] An increase in frequency during cardiac operations is not surprising.

Outside the operating room, cardiac arrest is well known in the recovery room, emergency room, endoscopy clinic, and radiology department, and its occurrence in any of these areas is associated with a poor prognosis.

Recognition of cardiac arrest is simple but deserves emphasis because of its urgency. Absence of pulsation in major arteries is all that is necessary to establish the diagnosis. Beck and others have repeatedly stated that more elaborate determinations only delay resuscitation.[1] Recently, monitoring of the pulse during operation has been advocated, and it seems likely that some simple monitor with a warning system will come into common if not routine use in the future.

Successful management of cardiac arrest requires perfusion of the tissue with oxygenated blood within four minutes of cessation of circulation. The initial problem is restoration of an airway and adequate ventilation. This can be done by mouth-to-mouth breathing, as recently advocated by Elam and Benson,[6] by mask breathing using an anesthesia machine, or preferably by endotracheal intubation. When good efforts to restore ventilation are begun, the managing physician should begin circulatory restoration by cardiac massage through the open chest. If operating in the abdomen, preliminary massage can be carried out through the intact diaphragm while the chest is being opened. It is important to emphasize to untrained persons how easily the heart itself may be cut in a too rapid thoracotomy. Massage is facilitated by opening the pericardium. The position of the surgeon during massage and the method of holding the heart are matters of individual preference. The possibility of myocardial injury or actual rupture from too vigorous massage should be mentioned. The initial rate of massage is 70 to 100 per minute, and the criteria of successful manual systole is the detection of a palpable peripheral pulse. Once massage is begun the requirements of the tissues for oxygen can be met and the problem becomes one of restoring a heart beat. After myocardial tone is regained, heart beat will usually begin spontaneously. Drugs are useful when myocardial tone does not respond to massage or when ineffective beat is initiated.

The appearance of ventricular fibrillation during cardiac resuscitation is common and not necessarily ominous. Defibrillation using an electric shock should be attempted after massage has restored good tone and color to the myocardium. If the usual single shock

at 120 volts (1.5-2 amp.) is ineffective, multiple shock and higher voltage should be used after intervening periods of massage. The value of high voltages, particularly in large hearts, has not been generally appreciated.

Several drugs may be helpful in cardiac resuscitation. Epinephrine is used to improve myocardial tone and conduction. It is injected directly into any accessible chamber of the heart. The use of epinephrine is not contraindicated in ventricular fibrillation. Calcium, as emphasized by Doctor Blalock, increases myocardial contractility, but may be contraindicated in patients who have received digitalis.[10]

Circulatory arrest is always accompanied by metabolic acidosis. As Campbell has pointed out, acidosis diminishes response of the heart to presser amines.[5] For this and other reasons, sodium bicarbonate should be injected intravenously in each case of cardiac arrest.

Isuprel may have a place in the treatment of intractable ventricular fibrillation.[13] Potassium, barium, procaine, and heparin have been advocated but at the present time their value is uncertain.

Other stimulant drugs appear to have little place in the treatment of cardiac arrest.

During cardiac resuscitation, several adjuvant procedures are useful.

The patient should be placed in the Trendelenberg position to aid in perfusing the central nervous system, as well as to aid venous return to the heart.

Compression of the thoracic aorta may be advantageous in increasing flow to the brain. This occlusion should be relieved at 15 minute intervals to avoid spinal cord injury.

The influence of coronary perfusion on cardiac arythmia is very impressive during procedures involving cardio-pulmonary bypass. Some form of aortic perfusion should be considered in the unusual instances in which it is feasible.

Cardiac pacemakers are being widely advertised as essential hospital equipment. Electronic pacemakers are effective in treating ventricular asystole due to a conduction defect as in surgical trauma to the Bundle of His or in the Stokes-Adams syndrome due to other causes. They will not initiate effective contractions in the usual case of cardiac arrest.[15]

Defibrillation of the heart can be accomplished through the intact chest wall using the method and apparatus described by Kouwenhoven and Knickerbocker.[11] This has been particularly useful in the cardiac catheterization laboratory where ventricular fibrillation is a definite hazard and where the diagnosis can be instantly made from the oscilloscopic monitor of the electrocardiogram. Closed chest defibrillation of the heart has also been successful after myocardial infraction and represents a real advance in the treatment of this form of cardiac arrest.

Recently, unpublished work by Kouwenhoven and Knickerbocker has shown that in the dog compression of the closed chest is effective in restoring circulation and arterial pressure up to 90 mm. hg. can be obtained.[12] This procedure was investigated as a possibility in lengthening the period of effectiveness of closed chest defibrillation but has clinical possibilities of its own. Doctor James Jude of the Johns Hopkins Hospital has now successfully restored heart beat in three patients with asystole using external compression of the chest.[9] This procedure cannot yet be placed in our permanent armamentarium, but it may find important uses in the future.

When the heart does not respond to resuscitative efforts, there is a difficult decision as to when to discontinue resuscitation. There can be no fixed rule in this regard, but it should be mentioned that recovery has followed periods of massage exceeding four hours. The length of time between arrest and massage and the presence of persistently dilated pupils are important factors in the decision to abandon resuscitation.

The over-all prognosis for permanent survival in cardiac arrest is exceedingly difficult to estimate, but the figure given by Stephenson is 29 per cent. There is no doubt that rapid recognition and prompt treatment will result in a high survival rate. In

most institutions considerable improvement in the present rate can be achieved.

The major cause of death after successful restoration of heart beat is injury to the central nervous system. The clinical picture is that of unconsciousness and progressive decerebrate rigidity ending either in death within hours or survival with neurologic damage. Very occasionally recovery follows definite evidence of brain injury. It has been suggested that death is caused by brain swelling, which occurs in response to the original anoxic injury. If this is true, prevention of brain swelling might allow survival in cases when the original injury is not too severe. A series from the Johns Hopkins Hospital has been reported in which patients with evidence of central nervous system injury after cardiac arrest were treated by inducing hypothermia with impressive results.[19,20] Spencer and Zimmerman have shown an increase in survival rate in animals subjected to hypothermia after circulatory arrest,[24] and unpublished data from the University of Oklahoma indicates that the brain does swell following circulatory arrest.[21] Work on the use of Urea after cardiac arrest has recently been presented by Javid at the Congress of the American College of Surgeons.[23] At the present time, the induction of moderate hypothermia, or the use of Urea, seems justified in patients who show evidence of central nervous system injury after cardiac resuscitation.

A final word on the role of the surgeon in the problem of cardiac arrest. The surgeon logically has the greatest experience and interest in this catastrophe, and it is urged that he assume a hospital wide responsibility for efforts to prevent, recognize, and treat cardiac arrest. Someone must emphasize the continued necessity for proper evaluation of pre-operative patients, the value of pre-operative medication, and the need for special anesthetic care and perhaps monitoring during some procedures. Someone must see that the equipment for resuscitation is available to remote areas. Most important, all physicians must be taught the principles of resuscitation and necessity for applying them promptly. The value of animal demonstrations cannot be over estimated. The salvage of 8,000 lives a year depends upon our efforts.

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A brief review of Ewing's Sarcoma and its incidence in the foot with two case reports involving the tarsal bones.

Ewing's Sarcoma of the Tarsal Bones— Two Case Reports

MARVIN K. MARGO, M.D.

JAMES N. OWENS, JR., M.D.

Marvin K. Margo, M.D., graduated from the University of Oklahoma School of Medicine where he is now Instructor in Orthopedic and Fracture Surgery. He is certified by the American Board of Orthopedic Surgery and is a Fellow in the American College of Surgeons, Fellow in the American Academy of Orthopedic Surgeons, the Clinical Orthopedic Society and the Association of Bone and Joint Surgeons.

James N. Owens, Jr., M.D., graduated from Vanderbilt University School of Medicine in 1938 and is now Assistant Professor of Pathology at the University of Oklahoma School of Medicine. He is certified by the American Board of Pathology and Anatomy and the American Board of Clinical Pathology.

Doctor Owens is a Fellow of the American College of Pathology, the American Association of Blood Banks, the American Society of Clinical Pathologists and the American College of Physicians.

ENDOTHELIOMA or Ewing's sarcoma of the bone is most commonly a disease of childhood with eighty per cent of all reported cases occurring under the age of thirty. There have been isolated cases reported in children as young as six months of age.

Ewing[1] reports that the small bones and flat bones are most often involved. Lichtenstein,[2] in his book of recent date, states that peripheral bones are most often involved. The etiology of Ewing's sarcoma to date is not clearly defined. Of the numerous reports of this disease in literature, very few have been noted in the tarsal bones.

Seventeen cases of Ewing's sarcoma are reported by Lichtenstein and Jaffe,[3] none involving the feet. Wang and Schutlz[4] reported 50 cases of Ewing's sarcoma, one being in a tarsal bone. McSwain, Bird and associates[5] reported 20 cases; none of these were in the feet. McCormack, Dockery and Ghormley,[6] in 1952, reported 80 cases of Ewing's sarcoma; one case involved the metatarsal bone. Of the 177 cases reported from the Mayo Clinic in 1950, none were reported in the small bones of the foot. Morton[7] reported 190 cases of Ewing's sarcoma in 1923; one case involved the os calcis and one a metatarsal bone. Frederick Christenson,[8] in 1925, reported 87 cases; four were in the os calcis and two in the metatarsal bone.

The above review reveals only 11 cases of primary Ewing's sarcoma involving the bones of the foot. Of these 11, five involved the os calcis, one of the talus, and four of the metatarsal bones. There is one article which refers to the location of Ewing's tumor in an unspecified tarsal bone. This report adds a case involving the talus and one of the os calcis.

The clinical course of the disease varies, of course, with the histological malignancy

and other factors involved. Some subjects succumb to a very rapid growth of the original tumor, while others have survived as long as nine or fifteen years. Mortality is very high, especially with the younger individuals. The roentgenographic appearance (onion peel) which has previously been reported, certainly is not borne out with our cases, nor has it been borne out with other writers who have referred to this disease. It is a tumor which arises in the marrow spaces of the affected bones. The typical cytological appearance is that of uniform cells with ill-defined borders, the cells having little cytoplasm. The nuclei, however, are indistinct at times and appear embedded in a very loose vacuolated cytoplasmic fabric. Most of the nuclei show scattered chromatin with the nucleus two or three times as great as the diameter of a normal lymphocyte. Marked vascularity and rosettes or pseudo rosettes, which have been described as being characteristic, certainly are not as frequent or as pathognomonic as has previously been reported. The appearance of rosettes and pseudo rosettes is altered most frequently by degeneration or necrosis of the tumor. One characteristic feature of this tumor, however, is the fact that no argyrophilic fibrils are to be found. The differential diagnosis of this disease should include metastatic carcinoma of hidden origin, neuroblastomas arising from either adrenal or sympathetic nervous system, myeloma, and lymphatic lymphoma.

Case Report I

D. W. is an 11 year old white male who was first seen on May 17, 1954, with the chief complaint of pain and swelling in the left ankle. He states that this condition has been present approximately three and one-half years, with intermittent pain and inability to walk at times. He had had numerous antibiotic shots and whirlpool treatments which gave no relief. His tonsils were removed at the age of four. Past history revealed he had chickenpox, mumps and measles when he was younger.

On initial examination he had a temperature of 99° and weighed 64 pounds. Physical examination was essentially negative except for the fact that the patient limped on



Figure I. Reproduction of roentgenogram of ankle showing sclerosis and mottling on the talus.

the left leg and there was visible swelling of the left ankle anterior to both malleoli. At this time fluid was removed from the ankle joint and this revealed only seven white blood cells per high power field. Cultures were negative both in aerobic and anaerobic media. Antistreptolysin blood titer was 166 Todd units. The sedimentation rate was 23 mm. in one hour on the Westergren scale, and he had a perfectly normal hemogram. Roentgenograms of the ankle at this time were interpreted as negative. He was checked at approximately one month intervals, and the left ankle at times was definitely swollen, very tender, and any motion of the ankle produced pain. There was no evidence of fluid obtainable on any subsequent examination. At frequent intervals he had a temperature which ranged from 99 to 102°F, although ENT examinations revealed no positive findings. On June 9, 1954, however, another aspiration of the ankle joint was done and five cc. of bloody dense fluid was obtained. The cultures and animal injections done at that time were en-

tirely negative. On October 11, 1954, the patient had developed increased pain with a mass appearing on the anterior medial aspect of the talus which was quite tender, and continued to enlarge since it was first noticed about three weeks previously. He was unable to walk on this foot due to increased pain; it was then that the biopsy of the tumor of the talus was carried out.

Roentgenograms taken at that time revealed some moth-eaten areas of decreased density as well as a few areas of periosteal proliferation on the neck of the talus. The body of the talus seemed to be well involved with the moth-eaten areas (Figure 1). The biopsy which was carried out under general anesthetic revealed a mass of pink, almost fish-flesh colored material which seemed to be beneath the ankle joint capsule. One opening the ankle joint capsule, the friable mass, which was 4 x 2½ cm. in size protruded into the wound. It seemed as though the entire talus was involved with this tumor. A posterior plaster splint was applied and a dry dressing applied. The father took the patient out of the hospital two days later and follow-up revealed that patient expired one year later with no autopsy being obtained.

Microscopic examination presented a uniform picture. The tissue is made up of sheets of dark blue staining cells approximately twice as large as an adult lymphocyte. The nuclei of the cells appear much larger than normal cells with very little accompanying cytoplasm. Only a few mitotic figures were to be seen. Numerous nucleoli as well as irregular strands of chromatin were in evidence. Many endothelial lined spaces were seen, which was taken to represent increased vascularity of the tumor. Tumor emboli were in evidence in several of the larger endothelial lined vessels, which appeared to be of arterial origin. Rosettes and pseudo rosettes were in evidence surrounding the larger blood vessels.

Case Report II

F.D.H.—The patient was an eight year old white male who was first seen on April 15, 1954, complaining of pain in the left heel which had been intermittent in character for a period of about one year. At the time of onset he gave a history of having gone down



Figure II. Reproduction of roentgenogram of ankle showing sclerosis and mottling of the os calcis.

a slide and injuring his heel. The patient had been seen by a doctor with a diagnosis of bruised heel but denied any laceration, contusion or abrasion. Roentgenograms taken at that time were entirely negative. He had had some night cries but these had never been with severe pain; this was usually relieved considerably by heat and to some extent by aspirin.

Past history revealed that he had had frequent infections of the tonsils during the previous two or three years, but he had been gaining weight and feeling very well with no other joints involved at that time.

Examination revealed that he was a well nourished, well developed white male who did not appear acutely ill and could walk. Examination was entirely negative except for the left foot and ankle where there was generalized swelling throughout the region of the left ankle and forefoot with pain on motion of the ankle in any direction, but especially on inversion. He had point tenderness on the lateral and posterior aspects of

the heel but there was no localized area of redness or heat. The roentgenograms were essentially negative. The sedimentation rate was 16 mm. in one hour on the Westergren scale; the hemogram was essentially normal, and the urinalysis was normal.

The patient was admitted to the hospital on May 4, 1954 due to an increase of pain in his foot which had required hypnotics at times for relief. His temperature had been normal. There was no lymphadenopathy. The foot was especially swollen about the heel with considerable tenderness on pressure about the heel. Antistreptolysin titer was 625 Todd units; the sedimentation rate was 40 mm. in one hour; the urinalysis was negative; the white count 5,450 with 50% neutrophiles, and 41% lymphocytes. Roentgenograms taken again were entirely negative. He was dismissed from the hospital four days later on May 8th. He was seen at frequent intervals and appeared to be doing very well other than the fact that there was a continuation of swelling in the foot and pain on motion; he was unable to bear weight on it. His temperature was 99.2°F. on several occasions. On July 28th, 1954 he was placed in a walking boot cast with very little relief. Roentgenograms made on August 25, 1954 revealed there was extensive mottling and a loss of normal bone detail in the entire os calcis (Figure 2). There was a thinning of the cortex of the body superiorly. He had had no febrile episodes. He was admitted to the hospital at this time. An electrocardiogram was negative. Anaerobic and aroebic cultures were negative. Cultures were done on Petragnani media which, after six weeks time was negative. He continued to run a temperature of 99 to 100°F. A biopsy was done on September 8, 1954 which revealed the bone and cortex to be of mushy-like consistency with a milky secretion on the under surface of the periosteum.

Microscopic examination of the tissue revealed that all normal histological features were destroyed. The tissue was replaced by cords and strands of basophilic, hyperchromatic cells, whose nuclei were enlarged and contained many nucleoli. Numerous mitotic figures were in evidence. Marked vascularity was to be noted with very scant stroma in evidence. Rosettes and pseudo rosettes were conspicuous by their absence.

Postoperative course following the biopsy was entirely normal and the patient was dismissed from the hospital to be given extensive radiation therapy, following which he developed numerous metastases to the chest and hips. The white blood count decreased to 3,000. The patient became blind in both eyes, and numerous pelvic, chest, and hip metastases occurred, and patient expired within a six months period of time.

Summary

A review of the literature has been made revealing only 11 cases of Ewing's sarcoma in the foot. Case reports of Ewing's tumor in the talus and os calcis bones of the foot have been added.

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J. M. Owens, Jr., M.D.
605 N.W. 10th, Oklahoma City, Oklahoma

ABSTRACTS

Wound Healing: Investigation of Proteins, Glycoproteins, and Lipids of Experimental Wound Fluid in the Dog

BETTY N. WHITE,* M. R. SHETLAR,** HELEN M. SHURLEY*** and JOHN A. SCHILLING.****

Proceedings of the Society for Experimental Biology and Medicine **101:** 353-356, 1959

Surgical implants of stainless steel mesh cylinders were made into subcutaneous tissue of dog's back. These were removed at intervals of one to four weeks. Wound fluid was aspirated from inside these cylinders encapsulated with fibrous tissue. When compared with animal's serum, this fluid was lower in protein, glycoprotein, and lipid; and these components decreased with time during post-wound period. As studied by paper electrophoresis, wound fluid had a higher per cent of albumin and lower alpha-2 and beta-1 globulin. An elevation of protein-bound hexose was found in beta and gamma globulin fractions at three to four weeks. Seromucoid was present at same level in wound fluid and serum. The lipid partition revealed especially low phosphatides but similar cholesterol: protein ratios. It appears that time-related changes occur in large molecular constituents of fluid found in wound area which may be significant in wound healing phenomenon.

*Research Technician, Department of Surgery.
**Associate Professor of Biochemistry.
***Research Technician, Department of Surgery.
****Professor of Surgery.

The Oral Yeast-Lactobacillus Relationship I. Nonutilization of Hexosephosphates by An Oral Lactobacillus

THOMAS E. WILSON,* PAUL W. GOAZ,** and HAL H. RAMSEY.***

The Journal of the American Dental Association **58:** 64-69, 1959

These studies demonstrate that an intact, viable, actively metabolizing oral strain of L. casei cannot, or at best can only very slowly and ineffectively, utilize glucose, 1-phosphate, glucose 6-phosphate, fructose 6-phosphate and fructose 1,6-diphosphate, for growth or lactic acid production. The results are also relatively unaltered by either the presence or absence of an oral C. albicans filtrate. The apparent synergistic activity of the yeast and the lactobacillus cannot be interpreted on the basis of a complementary effect of certain glycolytic enzymes from each microorganism (as suggested by the earlier workers) inasmuch as the first and major premise of this theory was speculation that the lactobacillus can readily utilize the hexosephosphates in the place of glucose as an energy source.

*Assistant Professor of Research Microbiology.
**Instructor in Research Dental Surgery.
***Research Corporation, New York.

Posthemorrhagic Anemia and Shock in the Newborn

HENRY N. KIRKMAN* and HARRIS D. RILEY, Jr.**

Pediatrics, **24:** 97-105, 1959

Acute fetal blood-loss usually occurs during labor and delivery. This condition may be the second commonest cause for anemia in infants at birth. Although the causes for fetal hemorrhage are diverse, they result in a single entity, posthemorrhagic anemia or shock immediately after birth. The signs and management are essentially the same regardless of cause. It is suggested that "posthemorrhagic anemia and shock in the newborn" be an accepted and standard pediatric diagnosis. The infants are pale but have no hepato-splenomegaly or edema. If the condition is severe the infants have weakness, tachycardia and distress out of proportion to any degree of cyanosis. Pallor persists in spite of administration of oxygen. Under such conditions immediate re-expansion of blood volume is imperative and life-saving.

*National Institutes of Health, presently Assistant Professor of Pediatrics Oklahoma University School of Medicine.
**Professor of Pediatrics.

Psychodynamic Factors in Munchausen's Syndrome

EVERETT R. RHODES* and ROBERT A. TOLAND.**

A.M.A. Archives of Internal Medicine, **104:** 427-429, 1959

A case of Munchausen's syndrome is presented in which it was possible to study the life history and (to a degree) the emotional development of the patient. In view of these data, the bizarre behavior of the patient takes on meaning and fits into a reasonable psychodynamic formulation. In this particular patient, we have seen a person who very early developed difficulty in establishing rapport with his environment. This

was followed by a series of incidents, each of which, in itself, was not unusual, but which, in this patient, added up to the bizarre behavior. It should not be presumed that these same mechanisms are operative in all cases of Munchausen's syndrome. Further study of patients with this syndrome is needed before drawing definite conclusions regarding the etiology.

*Second Year Resident in Medicine.
**Director, Brazos County Youth Development Service, Bryan, Texas.

Posthemorrhagic Anemia and Shock in the Newborn Due to Hemorrhage During Delivery

HENRY N. KIRKMAN* and HARRIS D. RILEY, Jr.**

Pediatrics, 24: 92-96, 1959

A comprehensive review of all aspects of posthemorrhagic anemia and shock in the newborn, based on the experience in this clinic and cases reported in the literature, has been provided in another paper by the authors.

Cases 1 and 2 represent examples of posthemorrhagic shock resulting from tearing of a velamentous insertion of the umbilical vessels. Case 3 is an example of fetal blood loss due to tearing of a vessel communicating between a succenturiate lobe and the main body of the placenta. Cases 4-7 in all probability represent examples of acute fetal blood-loss due to incision or disruption of the placenta. Although the source of bleeding was not recorded for Cases 2 or 4-7, a posthemorrhagic state could be presumed by the condition of the infants and the coexistence of obstetric conditions with which fetal bleeding is commonly associated. Case 8 is an example of acute fetal blood-loss due to disruption of the placenta after uterine rupture.

*National Institutes of Health.
**Professor of Pediatrics

General Principles in Treatment Of Injuries to Athletes

DON H. O'DONOGHUE*

The Journal of the AMA, 171: 1656-1659, November 1959

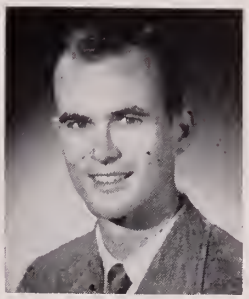
Experience has led to the conviction that a physician called on to treat an injured athlete should respect the patient's desire for early return to a competitive status. The physician's examination should be thorough, and treatment from the beginning must aim at the complete rehabilitation of the athlete.

*Professor of Orthopedic and Fracture Surgery.

New Faculty Appointments

Recent appointments to the School of Medicine faculty include new members of the departments of Medicine, Pediatrics, Anesthesiology and Preventive Medicine and Public Health.

Newcomers to the Department of Medicine are Stephen Wright Thompson, M.D., and Natoo C. Patel, M.D., instructors.


THOMPSON

Doctor Thompson, who received his M.D. in 1951 from Ohio State University, interned at University Hospitals and served an internal medicine residency at the Medical Center, then took a residency at Baylor and returned here last year for further residency work in neurology at the Oklahoma City Veterans Hospital.

He is doing research on the determination of regional blood flow by surface counting of radioactive tracers.

Doctor Patel, a native of Bombay, India, came to the Center from the University of Colorado Medical school where he was chief resident in medicine the past two years. A graduate of Podan Medical College, Bombay, he subsequently received a master of science degree at the University of Colorado. He has taken additional residency training in medicine, pathology and cardiology.

Named to the Department of Pediatrics were George Bouthilet, research associate, and Leon Horowitz, M.D., Tulsa, visiting lecturer.

Joseph Louis Martin, M.D., is a new clinical assistant in the Department of Anesthesiology; and Everett Neal Holden, M.D., in the Department of Preventive Medicine and Public Health.

Special Articles

PROFESSION OR BUSINESS?*

J. H. MEANS, M.D.,** Boston

MEDICINE, traditionally, has been considered to be a profession, even one of the so-called learned professions. Many definitions of profession can be found, but the idea common to them all appears to be that a profession is organized for service to humanity, whereas a business or trade is entered into for the sake of material profit. At first blush it might seem that there should be no confusion or overlapping between these, and I believe that until quite recently there has not been. The medical profession especially has valiantly put in writing its concepts of what constitutes proper professional behavior for the doctor of medicine. The late Willard L. Sperry, D.D., in his important little book entitled, "The Ethical Basis of Medical Practice," has this to say:

> Of all professional groups that of medicine has, on the whole, codified its ethics more rigorously than has any other of the kindred professions. Lawyers and teachers have their codes of ethical practice. In the former instance they are probably less rigorous, and in the latter instance more vague than in the case of medicine. As for my own profession, that of the ministry, unofficial codes of ethics are as numerous as the leaves of Vallombrosa, but they lack the authority of these other codes.

But as after Moses brought the Decalogue from God to man, lapses into the worship of Mammon came with increasing frequency, so also medical practice is presently slipping more and more from its high ethic into the behavior pattern of the market place. A nonmedical writer within a few months has even had the temerity to bring out a book entitled, "The Doctor Business." It is an item that is causing some disputation among reviewers. I believe, therefore, that it is appropriate in the Gay Lecture this year to examine critically to what extent and in what direction deterioration is occuring in the boundary that should separate profession from business in the realm of medicine.

Ethics

Let us begin with a look at some of the existing codes of ethics. The oldest and most famous in our vision of history is the Oath of Hippocrates. I assume that you all know it by heart. There were others before it among the Egyptians, Sumerians and Babylonians. But for us the take-off is customarily from Hippocrates. The significance of

*The George W. Gay Lecture, delivered at Harvard Medical School, May 13, 1959.

**Jackson Professor of Clinical Medicine, Emeritus, Harvard Medical School; honorary physician, Massachusetts General Hospital.

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the Oath of Hippocrates, composed in the fifth century B.C., is that in its major principles and in its spirit, it is perfectly valid today. It is a noble utterance. To be sure, Hippocrates swears by Apollo and some other deities, which we don't have to do, but recall that Jesus told us not to swear at all —merely to tell the truth. So we are free to affirm the substance of the Hippocratic Oath, and what's more, to govern our professional lives by it.

The "Principles of Medical Ethics" promulgated by the American Medical Association, as far as it goes, is also an impeccable document. I can subscribe to nearly all of it. It has been through various versions. In the last one—1958—it has been reduced to a decalogue, almost as pithy as that of Moses. You should read it. The first paragraph of an earlier version of the "Principles" contains a statement that I like especially: "The practice of medicine is a profession. In choosing this profession an individual assumes an obligation to conduct himself in accord with its ideals." Some of the state medical societies have codes of ethics antedating that of the AMA.

Now, however, in 1959, we find ourselves in situations that make full compliance with the traditional ethical standards of medicine ever more difficult. Society has been overtaken by a socioeconomic turmoil with various innovations and reactions. These create all manner of new pressures, which push the doctor about until he is fuzzy enough not to be able always to distinguish between the ethical and the unethical. On all sides he sees business, unhampered by any of the principles by which he himself is bound, getting progressively more aggressive in the pursuit of profits through ruthless competition. He sees predatory advertising invading more and more systems of the social body—transportation, roadside and all the mass media. He sees chiselers and racketeers boring through the otherwise healthy social organism in a great variety of directions. He himself is forbidden by all his ethical tradition to engage in advertising, or to solicit patients, which amounts to the same thing, but he has to live and function in a culture ridden by advertising. Indeed, the function he serves is a vital part of that culture. Under these circumstances it is not surprising to find him sometimes making departures from his traditional pattern of behavior, and this he does both as an individual and through his professional organizations.

Nothing puts more strain on the doctor's ethics than financial matters. You may recall that St. Paul assures us that love of money is the root of all evil, and we all know that the more competitive a society, the more the power of money will be felt. Two of the new pressures that impinge on the doctor arise from the problem of how he shall be paid for his services and how, or in what manner, his services shall be supplied.

The traditional method of paying the doctor has been by fee for each service as rendered. When a patient could not pay the fee, the doctor nobly reduced it or waived it altogether. In an older, slower moving and less specialized society, this method has been satisfactory both to doctors and to patients. When practiced honorably it is certainly above reproach from the point of view of ethics. But that is not to say that in present-day society it is necessarily the *best* way, although it still seems to be so regarded by many doctors. But from the patient's side there is steadily increasing dissatisfaction with it.

A considerable number of objections to fee-for-service solo practice of medicine can easily be raised. It lends itself to abuse in various ways—fee splitting, for example. This is an ancient sin of medicine that is still with us, but I mention it in the present connection merely to illustrate again the ethical difference between profession and business. There is nothing unethical about the equivalent of fee splitting in business according to the ethical standards of business. It is merely granting a commision to someone who brings in more business. If a professional person, however, engages in such practices, it lowers his standards to those of persons who do business for profit. It puts the fee splitter in competition with other doctors and to trafficking with human life, health and happiness. Among other abuses we have such things as the rendering of a

service and charging a fee for it, when the doctor, although the patient may not know it, is not really competent to perform it. Even in Hippocrate's day this problem must have been rife, for he said, "I will not cut for the stone but leave that to practitioners of that work." On fee-for-service there is also the temptation to do unnecessary surgery or provide other forms of unneeded treatment to increase the income from fees.

From the patient's side, fee-for-service also may have the ill-effect of keeping patients away from the doctor when they really need his care, just because they wish to avoid the paying of fees. Indeed, the early diagnosis of disease and the whole matter of preventive medicine may become obstructed by the fee-for-service system. On prepayment, on the other hand, these services are included in the comprehensive program and create no such difficulty. On fee-for-service a conscientious doctor may be sore perplexed to draw the line between what is proper safeguarding of his patients by calling them in for check-ups, and what is exploiting them by insisting on unnecessary visits.

The progressive fragmentation of medicine into ever-narrower specialties also makes the furnishing of comprehensive medical care on the fee-for-service solo-practice basis increasingly difficult. When one doctor could do nearly everything and people for the most part stayed put for long periods, this was quite easy. The day when that sort of thing was possible, however, is past. Although one generalist can still do much of it, he will frequently need help from specialists, often several in a single case, and when all these bill the patient independently, it imposes a quite unnecessary hardship on him. It has driven some patients to go on their own initiative directly to specialists, very probably the wrong ones, without either patient or doctor knowing that they are the wrong ones. Not infrequently nowadays patients engage in what is known as medical shopping. They make up a list of specialists of their own choosing, quite probably on the basis of information from friends, and consult as many of them as they think indicated. They may think that in this way they are getting adequate medical care, but they are not. Some of their parts may be cared for, but not they themselves. The specialists involved may not even have any communication one with another about the patient's problem, and in any event no one doctor is taking the responsibility of integrating the total care of the patient. Only an understanding personal physician can do that adequately.

It can be said, indeed emphatically, that one of the major ethical principles of medical practice is that the doctor must be alert enough to perceive his total responsibility in every case and have the determination to meet it fully. His responsibility even continues after death, because when a patient of his dies, it is his duty to make every effort to obtain an autopsy. The reasons for this are both scientific and moral. Scientifically, it is valuable always to have autopsies because clinical impressions will be checked against indisputable material evidence, and because there is always the chance that something new will be learned. It is morally important to have an autopsy because it takes courage on the part of the doctor to face one. The knowledge that he will have to do this puts him on his mettle always to give the best care he can. It is not good medicine to bury one's mistakes, sight unseen.

When skills other than his own are needed in the care of his patient, the doctor has the responsibility of obtaining the aid of others who can supply them. That is to say, he must call in consultants. Any reluctance to do this is unethical.

It is not enough, however, for doctors either as individuals or collectively, merely to avoid being unethical, to stay only within the letter, but not the spirit of the ethical code. "Such ethics," a philosopher, A. E. Meyer, has recently said, "should rather be called practical rules to get along in life; they are selfishness brought into harmony with the demands of others, who claim equal rights and are willing to enforce them. It is the kind of ethics that has presented itself in international politics, demanding a world organization to prevent future wars."

Is the medical profession today morally bound to go beyond this sort of thing and voluntarily take certain positive actions to

improve the provision of medical care in the country at large? If so, is it doing this? Of course, these questions can be evaded by asking whether there is really any need or room for improvement in the medical-care establishment of the United States. I hope that what I have already said will have convinced you that there is—from the point of view not only of getting integration of divers skills in giving care but also in putting payment for care on a basis financially fairer and more equitable to patients than the presently prevailing system of solo practice and fee-for-service, with free choice of doctor.

If actually there is room for improvement, what line shall it take? Probably there are several possible ways, and the indication is to try some of them out and discover what they have to offer. This actually is already being done in a somewhat spotty fashion, but only with vigorous opposition from organized medicine. A minority of the profession, but one growing in size, believes that the provision of medical care largely by groups of doctors, organized to include a proper proportion of generalists and specialists, and paid for their services on a prepayment plan of some sort, offers many advantages to both patients and doctors over the present system.

It is not my purpose, however, to discuss the pros and cons of any plans for the improvement of medical care at this time, and I am sure it would be unethical for me, within the framework of a Gay Lecture, to try to persuade you to take a particular side in a controversial matter. I wish merely to indicate the ethical principles that I believe to be involved. This can readily be done by asking the rhetorical question, What kind of ethics is it when organized medicine resorts to boycotting, lobbying, and other pressure tactics when honest attempts are made to improve medical care in the organizational and payment realms of medicine? What kind of ethics is it also when a county medical society not only refuses to co-operate with a group health plan being set up in its area by doctors of competence and integrity but instead does its utmost to scuttle it? In such a case the health plan bids fair to raise the quality of medical care and make it more generally available. The people want it, but the medical society fights it just the same. This sort of thing happens repeatedly. Such self-protective behavior is expected in business. It has no place in professional work of high ethical character.

The distinguished jurist, Mr. Justice Frankfurter, who gave the Gay Lecture a year ago said, "My profession is pretty bad . . . but I am bound to say that I should be a little troubled if my profession lined up an advertising agency to work out its relations with the government." This refers to the episode in 1950 during the Truman administration when the officials of the AMA raised an enormous "War Chest" to fight what they called "Socialized Medicine" and hired the advertising firm of Whitaker and Baxter to do it for them. They were for the moment successful, but at the cost of adopting behavior patterns more suitable to business or politics. The point I should like to make is that organized medicine should abide corporately by its code of ethics as assiduously as it expects its individual members to do. Any double standard in this regard is intolerable.

Thus far we have been considering ethics largely in relation to the care of patients, or practice of medicine in the usual sense, but medicine in its entirety encompasses much more than that, and ethics is involved also in these wider reaches. May I again recall the wisdom of Frankfurter. "I am not competent," said he, "to have any views on the very difficult problem of the relations between the medical profession and society. But the notion that it is just an individual affair between a patient and a doctor seems to be totally discredited by all we know." Justice Frankfurter has indeed here given us a strong hint of the multidimensional nature of our professional commitment, which, if heeded, cannot but intensify our interest and deepen our understanding both of our obligations and of our opportunities. Doctors, particularly organized doctors, are inclined to entertain the naïve belief that they are quite competent to appraise objectively their own profession. But since medicine is one of the vital functions of the social organism, of concern to all its members, this professional insularity has no valid vindica-

tion. It is significant, I think, that in the long list of Gay Lecturers we find a goodly number of disciplines other than medicine represented. This has been particularly true in recent years. We are getting, it seems, increasingly willing to see ourselves as others see us. Indeed, starting from today, we have to go back five years to find another physician as Lecturer. A lawyer, a psychoanalyst, two parsons and a writer intervene. This is all to the good.

This vista discloses another great ethical principle — namely, that medicine is for everyone. It is not a private game preserve for doctors. And how much greater is the happiness to be got from it if it is practiced in a generous spirit! Where doceors develop a proprietary interest in their patients, as believe me they sometimes do, they downgrade medicine to the level of business.

Medical Education

Let us now explore for a moment another great function of medicine—namely, teaching—and see what ethical principles may be involved in it. The medical profession is obligated to educate its own successors. Should it not do this the profession would die, for no one other than doctors of medicine can complete the education of doctors of medicine. The first stipulation in the Oath of Hippocrates is "to reckon him who taught me this art equally dear to me as my parents, to share my substance with him and relieve his necessities if required; to regard his offspring as on the same footing with my own brothers, and to teach them this art if they should wish to learn it, without fee or stipulation, and that by precept, lecture, and every other mode of instruction, I will impart a knowledge of the art to my own sons and to those of my teachers, and disciples bound by oath and stipulation, according to the law of medicine, but to none others." In modern parlance I think we may interpret this to mean that we have the obligation to teach, whenever the opportunity presents, anyone rightfully involved in the medical educational process if he so desires it.

Not only have we this teaching obligation, but if we are in medicine because we love it, we should desire to do it. We should derive happiness from it. The relation between teacher and pupil can become as meaningful and endearing as that between doctor and patient. And the medical teacher is challenged above all others because he has to meet the needs at the same time of both pupil and patient when the latter is the subject of instruction, nor can he allow the best interest of the one in any way to infringe upon that of the other.

Another point is well illustrated by the oft quoted lines of Chaucer, "and gladly would he learn and gladly teach." Where can one find a nobler precept for medicine than this? It puts learning before teaching, and how wise this is, because teaching at its best is no more than the facilitation of learning. I was extraordinarily happy three years ago at being enrolled a "Perpetual Student of the Medical College of St. Bartholomew's Hospital" (London). The word perpetual is the significant one in this charming epithet. It implies, or at least expresses, the hope that one may continue to be able to learn as long as he draws breath. So I pray may it be with all of us.

It would not seem likely, offhand, that there had ever been fee-for-service problems about paying for instruction by students, comparable to that for medical care by patients, but such has indeed been the case. Of course, in the days when medical schools were not available to all wishing to study medicine, there was the apprentice system, but that is not what I have in mind at the moment. In the eighteenth and early nineteenth centuries, students in medical schools paid fees for instruction directly to their professors—a fee for each course of instruction. The great John Hunter's fatal attack of angina pectoris was induced by a quarrel with other members of the staff of St. George's Hospital over a problem of fees paid by students to teachers.

At Harvard also in the eighteen forties, students paid fees directly to their professors for courses of lectures and were given tickets of admission. For the course in anatomy and operative surgery by Doctor J. C. Warren they paid $15, and for that on materia medica by Doctor Jacob Bigelow the fee was $10 and so forth. But all this is long

past history except perhaps in the case of certain graduate courses. Therefore, we may ask, if a better way than fee-for-service has been found for paying for instruction, why should not a better way than this be found also for paying for medical care?

We have today, moreover, an ethical problem concerning medical education at the collective level. The population, as you well know, is increasing at an explosive rate, and the need for well trained doctors is shooting upward. Medical education is the most costly of all education, and our medical schools are sorely pressed financially. No adequate aid is in sight short of federal subsidy. Yet organized medicine is so hostile to any further extension of government into medicine that it has opposed vigorously any help by the federal Government toward cost of instruction in medical schools.

Research

The second precept of the AMA decalogue tells us that "Physicians should strive continually to improve medical knowledge and skill, and should make available to their patients and colleagues the benefits of their professional attainments." Certainly, we can all say, "Amen" to this. The chief instrument to improve medical knowledge and skill is research, and this is another of the great functions of medicine. If without education we have no new doctors, so without research we have no progress. If we can say, "and gladly teach," so too we must say, "and zealously investigate." There are some ethical considerations about research that need to be mentioned.

The first of these is that the investigator must be scrupulously honest in all he does, both intellectually and in every other way. Complete objectivity is an absolute necessity. One should undertake research to test hypotheses, not to prove them. The object is to find the truth without bias or slanting of data. When a research is undertaken to discover whether cigarette smoking causes cancer of the lung, and is financed by tobacco interests, its results cannot but be suspect. Great temptations lie in the path of the investigator. Let us suppose he discovers something of importance—perhaps a new form of therapy. He tries it out on patients (after having done so first on animals). Its administration is followed by improvement in some of them. In reporting his results he is under great emotional pressure to see the good results through rosy, and the bad through dark glasses, or to classify as good that which is actually equivocal. Results of research seemingly important should not be accepted as fully valid unless confirmed by other investigators. Never forget that in research adequate control of all observations and experiments is mandatory, and that in clinical research on patients it is sometimes very difficult to achieve.

Another temptation is to utilize research for purposes of personal advantage or advancement. This really is a tough one because the habits of faculties are such that a regrettable amount of weight is put on how much a man has published. It would hardly be human not to like success, recognition and fame in research, but these should not be ends in themselves, but merely pleasant by-products. Nor should we in medicine keep our findings up our sleeve, as does business its trade secrets. At all stages of investigation we must be willing to exchange information with colleagues. This precept is indeed directly implied in the AMA's "Principles" (Section 2). Priority is dear to the hearts of all investigators, and why not? It is very commendable to be the first to uncover some new truth, and one may get a Nobel Prize for it, but the goal should be the new truth, not the kudos of priority. Moreover, in all research conducted in the professional spirit, investigators should give freely and generously full credit to other investigators whose work is in any way relevant to their own. To repeat the work of others without prejudice and publish findings with complete honesty is right and proper, indeed necessary. To belittle it in order to build up your own is, in the professional way of life, ignoble. In business, on the other hand, it seems to be rather normal, or at least frequent. Actually, to most discoveries many investigators contribute, and all deserve credit for what they have done.

Pharmaceutical Advertising

Increasingly in these days medicine is being confronted with difficult questions in ethics through the impact of the pharmaceutical industry. Its advertising infiltrates our medical journals, and many of them could not survive without the income it brings in. Practitioners receive daily through the mail vast quantities of multicolored beguiling advertising literature, well laced with free samples of decorative and expensive pharmaceutical preparations with trade names often difficult of comprehension, and complete with directions how to use them and with hand-picked bibliographies. For the doctor to prescribe such material solely on the recommendation of the manufacturer is to slight, in some measure, his duty by his patient. The rightful way for him to order drugs is to discover, from objective sources, their composition and pharmacologic action, and order them only to meet indications for treatment that he clearly recognizes. Then there are the detail men, usually delightful and co-operative fellows, who give you more advice and free samples, which, if you accept them, influence you somewhat to favor the detail man's house in your prescription writing.

The other side of the picture, however, is that the more reputable drug houses have made great strides in raising their ethical standards, and have done invaluable work in devising and testing new drugs in their research departments. They can get high-grade investigators because they can pay better salaries than universities or noncommercial research institutes. Furthermore, so lucrative has the pharmaceutical industry become, and so enlightened, that it makes larger and larger grants-in-aid to noncommercial research projects. Thus, even the universities become beholden to some extent to the industry. How good this is I confess I don't know, but I am strongly of the belief that it is necessary for medicine to work toward some form of ethical symbiosis with the pharmaceutical industry, if this can be done without compromising our own ethical standards. This is necessary because the industry can produce new scientific knowledge that medicine simply cannot afford to do without.

Patents

And this brings us to a special subject that sharply emphasizes the difference between business and profession—namely, patents. If a medical investigator develops a new drug or method, he is ethically bound to tell the professional world about it. He is prevented by his own ethical standards from patenting it. But if a drug house does such a thing, it would be, by business standards, insane not to apply immediately for a patent. But unless a discovery of noncommercial research is protected in some way, industry may patent it and make profits out of something to the discovery of which it has not contributed. Some ways have been found to escape from this dilemma. Patents can be taken out to protect a product, but dedicated to the public. This ensures that no one can gain a monopoly in its production. A well known example was insulin, which was patented by the University of Toronto to ensure its availability to all without anyone having any special privileges.

The Press

Another somewhat special topic is the press and other mass media. In the past the doctor has been supposed to keep himself out of the press as far as possible, at least not to use it as a means of popularizing himself. But nowadays when he cares for VIPs, he cannot keep himself out of the mass media, and this through no fault of his own. Also, often he is pushed into public writing or speaking by the institutions with which he is associated—hospitals, medical schools and so forth, which have money-raising campaigns under way and want him to glamorize what they are doing for the advancement of medical science.

All the mass media also carry a steadily increasing volume of medical items, purportedly for purposes of public education. Some of these are excellent; some are dreadful. The great question is whether the total impact on the public of all this is good or bad. When it provides the stimulus that brings the patient to the doctor while his serious

disease is still curable, it is good. When it leads to the pressurizing of doctors by patients to give treatment the doctor knows is not really indicated, it is bad. It has come to pass nowadays that patients, having soaked up some of the mass-media pronouncements, go to the doctor and tell him how to treat them. "Doc, I want a shot of penicillin." The doctor may know that this is neither needed nor desirable, but he also knows that if he doesn't give it, the patient will be off to another doctor who will. So the first doctor may give it even against his best judgment. Actually, he is on a hot spot. Of course in this case the ethics is clear enough. He should refuse to give it even though it means the loss of a patient. If he were not on fee-for-service there would be less of a problem.

The mass media can be very damaging also to the investigator. They will quote him out of context, give undue emphasis to parts of his remarks, and let headline writers commit veritable mayhem on what he has actually said: "Great breakthrough in cancer just around the Corner," and all that sort of rubbish. Beware of them!

The Patient and the Truth

A very controversial ethical question is that of truth telling to patients. You will find entirely reputable and earnest doctors who believe in lying to patients when it is, in their opinion, to the patient's good. The other extreme is best typified by the late Doctor Richard C. Cabot, of Harvard, who believed that the doctor should always tell the patient the truth, the whole truth, and nothing but the truth. This is a naïve position, because who ever knows, in the progress of a patient, what the whole truth is? I am sure some of the "truth" that Doctor Cabot told just wasn't so. For him things were either black or white, but the world is mostly gray. Personally, I am thoroughly opposed to the intentional practice of deceit in doctor-patient relations, but also I have constantly in mind the admonition of Oliver Cromwell, who said, "by the bowels of Christ my brethren bethink you, you may be mistaken." Also I cherish the dictum of the late Lawrence J. Henderson, "in the practice of medicine do as little harm with words as possible." Thus, I would say, beware of making traumatic remarks within the hearing of patients that they have neither asked for nor need to know. I am sure that this is one of the commonest professional sins that any of us commit. Beware particularly of ever making a prediction of how long anyone is going to live. Nature is capable of letting you down badly. Many a patient has outlived the doctor who "gave him but a year to live." Remember always that it is not what you say to patients that is important, but what they think you said. Your silences also may be as traumatic as your words. How often does a patient when asked, "What did the doctor tell you?" reply, "He didn't tell me anything!" The wise doctor will sense when the patient needs to be told something, and he will do this in as reassuring a way as the basic framework of truthfulness permits. Do not be afraid of saying, "I don't know," when that is the truth. Moreover, always be ready to listen, and to answer questions as wisely as you can.

Related to what you shall say, or not say, to the patient is what you shall say, or not say, about him to other people. Hippocrates puts us straight on this without need of alteration or interpretation: "Whatever, in connection with my professional practice, or not in connection with it, I may see or hear in the lives of men which ought not to be spoken, I will not divulge, as reckoning that such should be kept secret."

Another ethical or moral question, quite shocking in nature, is what we shall do about the elderly or hopelessly ill patient who longs for an end to his suffering in death. Shall we let him die, merely easing symptoms as he goes, or shall we force him to suffer yet a little while longer because science has given us such things as antibiotics and oxygen tents that will do this. Euthanasia, as you well know, is a hotly debated matter. Whether it may or may not be practiced is for society, not the medical profession, to decide. It is within the doctor's prerogative, however, to determine what treatment he will or will not use, and if under the type of circumstance I have indicated, he elects not to use the life-prolonging methods, can any honest and reasonable person say he has done

wrong? "Lord, now lettest thou thy servant depart in peace, according to thy word," so said St. Luke, "the beloved physician," and he expresses my own thoughts on the subject perfectly.

Discussion

I will finish this discourse with a few more transcendent considerations. Medical ethics is, I suppose, a part of ethics, and ethics certainly is a branch of philosophy; therefore, we may philosophize a bit. I am sure few, if any of us, are philosophers, but it is given to any man to apply his own homely philosophy to what he is doing and see what he can come up with in the way of convictions, motives and satisfactions. Recognition of these qualities of mind comes through insight, and certainly no one needs insight more than the physician. "Physician, know thyself" is perhaps the simplest way to put it. Although in a profession, especially in those called learned, of which medicine is one, the less conformity is required the better, nevertheless when in the practice of such a profession the lives, welfare or happiness of other people are involved, it becomes necessary for that profession to impose upon itself, or when it fails to do this for society to impose upon it, certain minimal standards of competence and certain rules of minimal acceptable professional behavior. It is these necessities that have given rise to the codes of medical ethics of which we have been speaking. I am convinced, however, that if a physician is of good conscience, there is little likelihood that his behavior will sink to a level where the ethical code need be called into play to correct it. At least this is true so long as he acts as an individual and follows the dictates of his conscience. If he has no consicence, he should not be in medicine. Unfortunately, as we have seen, when he organizes and acts collectively, the conscience of the herd, through a homogenizing process, may become sufficiently benumbed to permit some deterioration in ethical standards.

The quality of medicine in the mass, however, will depend largely on the motivation and character of the individuals who serve it, and the chief point I want to make is that the highest motivation possible is that one shall love medicine and find happiness in performing any of its many functions. This is indeed the essence of professional motivation and of what determines the professional way of life. It is what sets the profession in a higher ethical bracket than occupations entered merely to make a living. To be sure, the doctor must have an income sufficient to permit him and his family to live comfortably and happily, and to have enough leisure to keep in close rapport with the culture in its various dimensions. There is usually no great difficulty about this. It is a setback for medicine, however, when the doctor permits himself to become intellectually and emotionally isolated.

As I look around at my many friends in medicine—those who are older (not so many of these now), contemporaries and the steadily growing number of younger ones, many of whom have been my pupils—I am increasingly convinced that those who get the most out of medicine, and more particularly those who give the most to it, are those who love it. Medicine is indeed a profession—not a business. Let us keep it so.

And in conclusion I again quote Hippocrates: "While I continue to keep this oath inviolate, may it be granted to me to enjoy life and the practice of the art, respected by all men at all times, but should I trespass and violate this oath, may the reverse be my lot."

Vicarious Wish Fulfillment for Young Boys and Old Boys

JOSEPH J. QUINN*

GEE! It's been three years this week since we rode in the cab of the Santa Fe Chief. Three years! But we shall never forget the thrill and the hospitality. So many people have asked us to rerun the article so that they, too, can partake of the unusual experience. Well, here it is:

Whooooooooooo! We were sounding the horn (siren) on the Texas Chief, crack southwestern limited, running between Chicago and Galveston.

Ever since boyhood days we have always wanted to ride in the cab of a locomotive. But when the locomotive went out, we knew we didn't have a chance to ride in the cab of a diesel streamliner. But we did—and how!

It all started back in August when we wrote a piece about the disappearance of the trackwalker. Robert F. Emmanuel, Division Passenger Agent of the Santa Fe, wrote us and explained that the trackwalker had disappeared because they now have a mechanical track detector. At the same time he invited us to visit the Santa Fe Operations Office in Arkansas City. Not being technical, we wrote in this column that we should prefer to ride in the cab of a streamliner.

Nothing happened for several weeks. Then Mr. C. W. Lane, Public Relations Director of the Santa Fe asked us to come see him. "So you want to ride in the cab of a streamliner? That might be arranged but it will take time. How would you like to ride the Texas Chief to Texas?" That suited us just fine. Lots of red tape. In fact, it's more dificult than to get a visa to behind the Iron Curtain. Nobody rides in an engine cab except top railroad officials and once in a great while a newspaperman.

Mr. Lane's assistant, Gilbert (Gil) Sweet, as fine a young man as you would ever want to know, helped make arrangements early last week. He was to escort us to Gainesville, Texas, and ride in the cab with us. Last Friday morning there were several railroad officials, including Mr. Emmanuel, at the station. We were introduced to everyone. The Texas Chief rolled into the station with 16 cars—a heavy load. We climbed that perpendicular ladder for 10 feet on the recessed steps. (That was the hardest part of the trip.) Then we met the engineer, E. J. Fitch of Arkansas City and the fireman (second engineer) J. R. Conrad, of Ark City. Also the road foreman of engines, W. H. Hoster of Newkirk. As only four persons are permitted to ride in a cab, Mr. Sweet rode in the lounge car.

The cab is a small place and it smells of oil and compressed air. We were taken back into the first unit and had the compressors, generators, etc., explained to us. Then came two sharp whistles. The units became noisy. The fireman shouted "Highball!" and we were off, sand on the spinning wheels.

Up there in the train's magic eye everything looked different. The engineer's cushioned seat is 11 feet above the ground. We stood by the window and watched the train roll, watched it gather speed. Traffic stopped at crossings as the train sang its song to keep out of the way. Then came Norman in a jiffy.

Down near Purcell Engineer Fitch said, "Wanna blow the horn?" "How many times?" we asked. "Make it four for this curve." We gave it two mediums, a short and then hung on. The engineer smiled and remarked "You oughta been a railroader." Then we asked "When are you going to hit ninety?" "Just a little way off," he said. We watched the needle. Seventy, eighty, eighty-

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five and then ninety for about three miles. The engine rocked and rolled at ninety—the speed limit. We were sorta happy when he pulled it back to 85.

All along the trip, back and forth to Gainesville, the engineer and fireman kept shouting "Clear block." That's when they saw the green light ahead. One would shout, the other salute and repeat. The same way with the speed signs. One would sing out "70" and the other would echo "70." The engineer was always busy, twisting throttles, testing the brakes, pulling the siren rope and holding his eye on the road like an eagle. He keeps his foot on the "Dead Man Control" all the time. If he takes it off for 10 seconds, the train comes to a shuddering stop. That's to protect against heart attacks, etc.

You can talk about your foliage tours but we had a breath-taking one going through the Arbuckle mountains. The diesels twisted and turned around cliffs where red sumac was clinging for life. The maples were crimson on the hills and the oaks hugged the hollows in yellow hues. Below, the river ran brown and tawny from morning rains. Intermittently the sky was cloudy, then blue, then gray. But the Arbuckles were a riot of colors that raced by like pieces of silk thrown high into a wind.

At Purcell we changed crews. Engineer E. R. Erneike of Cleburne and Fireman J. C. Cox of Cleburne took charge. But we also had a V.I.P. on board. He called our name; knew all about us. He was O. D. Crill of Fort Worth, Superintendent of the northern division of the Santa Fe. He has his own special car, secretary and chef besides his Fort Worth office. But he rode to Gainesville with us in the cab and was a boon companion. Down across the Red River, we opened up to 80 through the plains. Then came to Gainesville. We climbed down, and were a dinner guest of Gil Sweet, together with Mr. Crill.

Two hours later we climbed into the cab again on the Texas Chief going north to Chicago. This diesel had a steamboat siren —and a loud one. Passing through stations, people put their fingers to their ears. The engineer was F. Webb of Gainesville and the fireman, L. H. Himmelreich of Cleburne. Mr. Crill also accompanied us in the cab back to Purcell where his private car was waiting.

Biggest Cinerama thrill of all was to cross a steel covered bridge going 80 miles an hour. You feel as if you will strike the supporting beams. Then you're over the river and the wooden ties appear like a black carpet unfolding before you. There seemingly is no space between them. We watched through the rear view mirrors and saw our end coaches swinging around the cliffs. When the last one was around the curves, the fireman would shout "All clear." Then the engineer would work the throttle and the train would lurch with gaining speed.

At Purcell we had the happiness of our first crew returning for their run to Arkansas City. Both Engineer Fitch and Fireman Conrad are the jolly type. They kept up a running fire of conversation.

Twice on the trip we held our breath. Down near Gainesville, a driver paused with his car on a country road. Then he stepped on the gas in order to beat us to the crossing. He escaped with his life by one second. The engineer shouted (because you have to shout in a diesel) "Some people want to be killed." Another time a huge gasoline truck was running fast for a crossing. Engineer Fitch touched the brake lever, and held the horn cord down, then shot single blasts, a dozen of them. The truck finally stopped four feet from the train. The engineer smiled and said to us "You came mighty close to being in heaven tonight."

Darkness fell at Norman. Coming into Oklahoma City the world was brown with dust. The panorama of the city at night is something to remember from a diesel cab. Red, yellow, green signs sparkled through the dusty air, trembling as if shaken by the wind. Skyscraper tops were lost, except for beacons. Then came the terrific hissing sound of the compressed air of applied brakes and the train glided like a jointed snake into the Santa Fe station. Our 280 mile trip was over—the most thrilling one we have ever taken.

Someone asked us if we wanted to do it all over again. Our answer was "No, sir, we have had our thrill and our fill."

Trends in the Distribution And Organization of Physicians In the Practice of Medicine*

L. W. MARTIN, Ph.D.

Assistant Director Economic Research, American Medical Association

THERE IS an old saying, "Two heads are better than one." An equally profound aphorism has it that "Too many cooks spoil the broth." These two observations underlie and help to explain much of the remarkable diversity which characterizes the organization of supply of goods and services in the United States. They relate in particular to the managerial, or more correctly entrepreneurial, conditions which affect the sizes of the business units in our economy. This point, I think, is quite pertinent to the organization of medical services in terms of the physician units supplying the services.

This is only one of many ways in which an economic analysis of the organizational arrangements for supplying medical services can be approached. Geographically the spatial arrangement and distribution of suppliers (physicians) may be related to that of the consumers of services, along with the communication and transportation patterns followed by these respective parties in achieving the provision of medical care.

Greatest emphasis, I believe, should be placed upon the diversity of organization which prevails and indeed thrives in the American economy. Spatially there are many different ways in which suppliers and consumers may be and are arranged in the American economy. The business organization is varied both among and within types. Additional elements of organization conceivably include the availability of ancillary facilities and services such as hospitals, laboratories, etc. The diverse methods of paying for medical care may also exert some influence. A much overlooked factor affecting industrial organization is the knowledge with which a good or service, as medical care, is consumed. The ramifications of the subject are manifold. It is not feasible to explore all of them in one brief talk; but at the same time silence should not be construed as ignorance of them.

There are certain broad questions to which I do not have answers and to which there may be no answers. For example, ought there to be more group clinics in the United States? Is a further increase in the number of group clinics likely? The first question is incapable of objective measurement; the second involves forecasting which can be done better the more one understands what conditions affect extension or contraction of this form of organization and the more knowledge he possesses on how these conditions are behaving. The common assumption that answers to these questions do exist in strictly objective, numerical terms, is, I fear, without foundation in fact.

The Oklahoma Situation

Group clinics are evidently more common in Oklahoma than in the United States generally. The available data are somewhat fragmentary and to some extent incomplete. Fortunately or unfortunately there is not one set of data; there are at least three. In all these sets presented in a table—with explanation of the ways in which the data are qualified by date and by definition—we can

*Presented at the Oklahoma Health and Welfare Association Meeting at the Biltmore Hotel, Oklahoma City, on November 16, 1959.

see that various rates, ratios, or proportions are higher in Oklahoma than they are in the United States generally.

Physicians, Group Clinics and Physicians in Group Clinics, Oklahoma and the United States

	United States	Oklahoma	Oklahoma/ United States
Physicians*	211,654	2170	1.03%
Group Clinics			
AAMC**	134	3	2.23
NACM+	433	9	2.08
AMA++	960	41	4.27
Physicians in Group Clinics			
AAMC	2,344	40	1.71
NACM	5,835	99	1.70
AMA	10,597	260	2.45
Physicians per Group Clinic			
AAMC	17+	13+	n.a.
NACM	13+	11	n.a.
AMA	11	6+	n.a.
% of Physicians in Group Clinics			
AAMC	1.1%	1.8%	n.a.
NACM	2.8%	4.6%	n.a.
AMA	5.0%	12.0%	n.a.

*American Medical Directory, 1958, Table 3, page 13. Government service physicians are not included in these figures. The effective date of these figures is approximately May, 1957.

**The American Association of Medical Clinics reported 134 members in 1959. This organization defines full membership as follows: Seven or more full-time physicians maintaining a private organization for the purpose of providing general medical care of high quality. The group shall have on its full-time staff at least five physicians in different major specialties, two of which shall be Internal Medicine and General Surgery. A group should maintain a separate building or group of offices for the conduct of its practice.

+The National Association of Clinic Managers reported 433 member groups in 1959. It defines membership as follows: An organization composed of 3 or more licensed doctors of medicine engaged in the practice of medicine and operating on a private basis with pooled facilities, coordinated laboratories, x-ray and allied departments for the purpose of diagnosis and treatment of diseases, and which has as its administrative head a full time clinic manager in active supervision of its business affairs.

++The American Medical Association's Council on Medical Service has almost completed a survey. Thus the figures we report from it have to be characterized as incomplete. For the purpose of the survey the group was defined as having 3 member physicians. The American Medical Association House of Delegates on December, 1948 defined group medical practice as follows: Group medical practice is the application of medical service by a number of physicians working in systematic association with the joint use of equipment and technical personnel and with centralized administration and financial organization.

The percentage Oklahoma physicians are of all U.S. physicians is exceeded by the percentage Oklahoma groups are of all U.S. groups and the percentage Oklahoma physicians in groups are of all U.S. physicians in groups. Moreover, a higher percentage of Oklahoma physicians are in groups than is the case for all U.S. physicians. The average size of groups in Oklahoma, however, is smaller than in the U.S. Roughly, Oklahoma has about 1% of the physicians in the United States, but, depending on the source one uses, it has either 2% or 4% of the clinics and from about 1.7% to 2½% of the physicians in clinics. Similarly, the proportion of Oklahoma's physicians in clinics is almost twice as high as that for the United States for all three sets of data available.

From a geographical point of view, the provision of medical care is oriented around the two big cities in the state, Oklahoma City and Tulsa. According to the study which Frank Dickinson made using 1950 population data and medical service rather than political areas as the basis of his analysis, these two cities, only 100 miles apart, comprised what he called prime-primary centers, of which there were then 88 in the United States. Within the state, he additionally found 28 primary centers of which 15 surrounded these two prime-primary centers. This area is suggestive of a broad axis running northeast or southwest parallel to a line connecting Oklahoma City and Tulsa. Whether there are more or fewer areas today or there could have been more or fewer in 1950, I cannot say. One wonders if a small primary area adjacent to a prime-primary area might have been merged with the larger unit.

Using the largest estimate of the number of group clinics in Oklahoma, 41, I find that 14 of them are in the Tulsa and Oklahoma City medical service areas and 17 more are in the 15 surrounding medical service areas. Thus I find 31 of the 41 groups in these 17 medical service areas. Three of the surrounding areas had no group clinics. Ten group clinics may be found in the 13 remaining areas, 4 of which had none and 1 of which, around the city of Ardmore, had 2. Just as there would seem to be a concentration of medical facilities and group clinics around Oklahoma City and Tulsa, approximately in the middle of the state and perhaps a little toward the northeast, the areas without group clinics comprised 4 medical service areas, 1 in the northwest, 2 in the southwest, and 1 in the southeastern parts on or near the state's borders.

Interpreting the Data

Interrelationships between the business and the spatial arrangements need to be in-

terpreted with great care. The presence of group clinics a short distance beyond the state's borders may help account for their absence—in a few cases—from the Oklahoma border areas. Moreover the concentration of group clinics in Oklahoma's big cities presents a pattern apparently different from that in the United States generally. According to a survey currently being made by the AMA's Council on Medical Service, almost a third of the groups identified are in cities under 10,000 in population and more than half are in cities under 25,000. Interpretations of the data available for Oklahoma may have to be confined to Oklahoma rather than extended to the U.S. Indeed, many factors ought to be considered in interpreting and evaluating trends in the distribution and organization of physicians in the practice of medicine.

In any interpretation of data on the organization of medical care services some further questions should also be asked. What advantages of size, for example, are there in the group clinic arrangement? How do groups adjust to changes in the realm of medical practice, changes primarily medical but having also economic implications? What external conditions affect the organization of medical services? In this last category I would include developments in transportation and in technical equipment, availability of ancillary facilities, and the knowledge which the consumer has and employs in his consumption of medical care.

Influencing Factors

Expectations regarding the growth and importance of group arrangement of physician units have often been exaggerated, I believe, because of exaggerated notions about the economies of size in our society generally. The production or technological advantages are usually exhausted in the single plant. Since all of our big firms are multiplant, their size is not required by technological conditions and their size has vastly exceeded the technological economies that can be derived.

In the area of decision-making, often called managerial but more accurtely entrepreneurial, the firms in our economy are characterized by much decentralization. General Motors copes with its size by having more than twenty substantially autonomous divisions, each of which is internally characterized by a great deal of managerial decentralization. Only in this way can the problems of coordination in a big organization be handled.

Much evidence is readily available on the diversity of American industry and the range in size of its firms. In autos, firms range in size from a small, single-plant parts supplier with a handful of employees to any of the big three. The flexibility and resiliency of a relatively small firm has recently been demonstrated by American Motors. Many of you are closely aware of the range of size in the petroleum industry from small well operators and amazingly small refiners to the biggest firms. Other industries as shoes and apparel are characterized by these variations in size. The bigger firms tend to make the more highly standardized, staple products, whereas the smaller companies make the more varied stylish specialties. The latter commonly require a lot of quick decision making. But this is not compatible with the big organization that tends to become afflicted with a great deal of bureaucratic red tape. Hence, my introductory use of the quotations and their relevance to the medical field where decisions usually should be made by one man, often should be made immediately upon the completion of the examination and the ensuing diagnoses, and less commonly require the advice of one or two additional men, but only rarely that of a great number of consultants.

Genuine economies of size probably lie in the areas of finance and marketing. The former refers to the ability to obtain funds. Big firms are usually deemed better credit risks and so can borrow at lower interest rates than smaller firms. They also can usually float capital stock. The big firm, moreover, can obtain a greater volume of advertising per dollar of sales than the small firm can. But the marketing advantage is of no consequence in medicine; advertising is unethical. Ordinarily the credit of physicians is very good, though it may well be that a group has an advantage over an individual physician in obtaining capital.

It may also be argued that a number of physicians will use capital (technical) equipment more intensively—and therefore more efficiently—than a physician practicing alone. I am much impressed by the combined facts that groups often have their own laboratories and sometimes hospitals and that they are more common in small than in big communities. One must wonder how much the formation of the group was due to the absence otherwise of ancillary facilities and the need to join together—possibly in order to obtain the capital—to provide these facilities. Surely any study of group practice should strive to study the presence or absence of ancillary facilities as a factor obviating or requiring the group arrangement.

The presence or absence of a need for group arrangements can be appraised in part by a review of other advantages often said to be associated with this arrangement —personal, professional, and economic advantages to the physician and the patient. Consider the economic advantages first. If the provision of physicians' services through the group arrangement is more efficient than individual practice, the physicians' incomes can be higher while fees to the patient are lower because the business operates on a lower ratio of expenses to gross receipts. But does this happen? Convincing data do not exist. The aforementioned AMA study currently in progress has not asked the question I have in mind; but the ratio of ancillary personnel to physicians has been checked. Since it is higher in the bigger groups, overhead is greater, and it is very doubtful that expenses comprise a lower proportion of gross receipts than in other arrangements.

The question of quality of care belongs to the medical profession and is beyond the competence of an economist. Physician-patient relationships are inviolate in all ethical forms of medical practice. Personal considerations for the physician can be cared for in a variety of ways. A physician practicing as an individual can make a working agreement with one or more other physicians. Likewise he can share facilities with him and possibly accrue some economies thereby. Again so many alternatives are available that compulsion toward one form of practice or another seems quite untenable.

My interest in the combined facts of the group's providing its own ancillary facilities and being more common in smaller cities is related to an important characteristic of economic development. This development in turn is related to the future prospects for group practice. In general as the market grows, smaller independent specialists appear to provide the ancillary functions which are usually shed by the older, bigger firms. If the need to provide its own ancillary services was an important factor in the formation of a group practice, this factor will tend to disappear with the growth of the community. The group may well continue in practice for any number of other reasons. New groups are less likely to appear in such a community (or region) though they may appear in other areas reaching a size sufficient to sustain a group but not as yet big enough to develop specialist ancillaries.

Consumer Knowledge

A final consideration relates to the knowledge with which the patient consumes medical, or physician, services. I am generally not in sympathy with those who belittle the rationality of the American consumer, though I do believe that the consumer is often uninformed. The glaring illustration of this, I believe, lies in the credit area of consumption where the ignorant consumer is victimized by excessively high carrying charges, interest rates and loan fees. Here as in some other respects consumer economics is a much neglected part of the subject.

The chief difficulty in the medical area lies in the consumer's assuming he knows what specialist to visit as soon as he is conscious of a symptom. In the medical area, as elsewhere in the economy, specialization or the division of labor genuinely contributes to economy, to efficiency, if properly employed. There has been some talk, justified or not, of overbalance toward specialization in the field of medicine, much of it suggesting that the responsibility lies largely upon the medical profession. If such an overbalance exists, the consumer undoubtedly exerts an influence in his quite commendable zeal for

the highest quality of medical care. He often thinks that a specialist will give him the best therapy whether his ailment is serious or slight and whether or not he knows exactly what his ailment is.

In the automobile repair field one does not consult an automatic transmission specialist for a squeaky clutch. Nor would it seem highly appropriate for one to visit an orthopedic surgeon for a twitch in the back, or to see a neuro-surgeon for a headache. These statements are exaggerated, of course. Our consumers ordinarily exhibit better knowledge and understanding of the pursuit of medical care than these extreme illustrations suggest. But ordinarily the manifestation of a symptom should influence a patient to call upon a general practitioner or, as some consumers now appear to insist, to visit an internist. Much time and other expense can be wasted in calling upon a specialist who proves not to be the one trained to care for the ailment ultimately diagnosed, with the result that the medical and economic benefits of the division of labor are lost. If knowledge and understanding of the desirable approach to the acquisition of medical care are lacking, the provision of medical care through a group arrangement may be more efficient than under individual physician arrangements. In this respect it would seem that some positive, constructive, educational work could be performed by consumer groups. I regret to say that, in contrast, certain self-designated consumer groups indulge in diatribes on medical care costs which often distort the data and misrepresent the facts.

There are a number of relevant factors which unfortunately I've not managed to discuss in this brief talk. Review of referral practices may expose a great deal of useful information about what physicians are doing and about some patient habits as well. Nor have I been able to raise the problem about what might occur in group practice arrangements when there ought to be fewer doctor members or, because of the progress of medical care or some change in consumer demand, the combination of generalists and specialists ought to be revised within the group.

I have not examined at all the future influence of changes in transportation and communication and their effects possibly or urbanization. If anything, I suspect that urbanization, of which suburbanization is a facet, will proceed in the coming years. I gather that via the turnpike one can travel from Oklahoma City to Tulsa in an hour and a half or less. The day may not be very far off when Oklahoma City and Tulsa will comprise one greater metropolitan area. Under these circumstances I would expect the division of labor to continue to grow—in medicine and its allied areas. What this may bode for group practice I surely cannot foretell for there are many other factors whose future developments and even the direction of them are much less easy to anticipate—and for any we may very well in the very near future be proved wrong in our present expectations.

Summary

Very briefly I have throughout this talk referred to the diversity of arrangements in our economy and the ways in which economic forces in our society apply to the practice of medicine as well as to the supply and demand of other goods and services. A few facts were cited on the geographical distribution of physicians and groups of them in Oklahoma as well as on the extent of group practice in Oklahoma. Some reference was made to the relationship between the situation in Oklahoma and that in all of the United States as well as to the implications of this relationship. Analysis of the factors affecting the presence or absence, extension or contraction of group practice casts doubt upon many economies claimed for group practice, points to the importance of better consumer education, emphasizes, once again, the variety or arrangements available in our society, and urges that studies of the distribution and organization of physicians—and other agents and facilities—in the provision of medical care consider the many variables mentioned in this talk as well as others presently beyond my limited imagination.

PRESIDENT'S LETTER



Oklahoma received more than usual attention at the Interim Meeting at the American Medical Association Convention in Dallas, Texas on December 1-4, 1959.

The award received by Doctor Chesley M. Martin of Elgin, Oklahoma was of national interest. The reasons he received this award might be considered well by all of us as objectives in our lives as physicians and citizens—selflessness and dedication. Incidentally, a member of the Award Committee told me that Doctor Martin was not only the outstanding candidate, but that the presentation, as prepared by our Don Blair, was the most outstanding in the history of the thirteen years of the award.

The Southwestern Hospitality Suite, sponsored by the Oklahoma State Medical Association and the associations of our sister states, was an outstanding success. The barbecued buffalo from Oklahoma was one of the main reasons. Great credit is due our delegates and alternates, who served as a committee in its planning.

Clyde T. Baker M.D.



Underweight Children Gain and Retain Weight with Nilevar®

One of the most convincing evidences of the anabolic activity of Nilevar, brand of norethandrolone, has been its ability to improve appetite and increase weight in poorly nourished, underweight children.

A highly important feature of the weight gain thus produced is that it is not ordinarily manifested by deposition of fat but as muscle tissue resulting from the protein anabolism induced by Nilevar.

Anorexia and "Weight Lag" Study—Brown, Libo and Nussbaum have reported* consistent and definite increases in rate of weight gain in eighty-six patients, ranging in age from 7 weeks to 15½ years. This beneficial action of Nilevar was observed in the patients with organic and traumatic disorders as well as those whose only complaints were poor appetite and/or persistent failure to gain weight.

In this study, the weight gained was not lost after discontinuance of Nilevar therapy although many patients did not continue the sharp gains effected by the drug.

The authors are of the opinion that Nilevar is a highly useful anabolic agent for influencing weight gain in underweight children.

When Nilevar is administered to children a dose of 0.25 mg. per pound of body weight is recommended and continuous dosage for more than three months is not recommended.

Nilevar is supplied as tablets of 10 mg., drops of 0.25 mg. per drop and ampuls of 25 mg. in 1 cc. of sesame oil. Further dosage information in Searle Reference Manual No. 4.

G. D. Searle & Co., Chicago 80, Illinois. Research in the Service of Medicine.

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*Brown, S. S.; Libo, H. W., and Nussboum, A. H.: Norethondrolone in the Successful Monagement of Anorexio ond "Weight Log" in Children, Scientific Exhibit presented ot the Annuol Meeting of the Americon Acodemy of Pediotrics, Chicogo, Oct. 20-23, 1958.

News

Actions of AMA Clinical Meeting

Freedom of choice of physician, relations between physicians and hospitals, a scholarship program for deserving medical students and relative value studies of medical services were among the major subjects acted upon by the House of Delegates at the American Medical Association's Thirteenth Clinical Meeting held December 1-4 in Dallas.

Oklahoman Receives Honor

Doctor Chesley M. Martin of Elgin, Oklahoma was named as the 1959 General Practitioner of the Year for his outstanding contributions to the health and civic affairs of his home community. Doctor Martin, who has practiced in Elgin for the past 44 years, was the 13th recipient of the annual award and the first Oklahoman to be so honored.

Speaking at the Tuesday opening session of the House, Doctor Louis M. Orr of Orlando, Fla., AMA President, urged the nation's physicians to take a more active interest in the whole area of politics, public affairs and community life. Doctor Orr also asked physicians and medical societies to do a more effective job of telling medicine's positive story, adding that "if more people knew more about the things we support and encourage, they would listen to us much more carefully about those occasional things that we oppose."

Two nationally known political leaders from Texas also addressed the Tuesday morning session. Senator Lyndon B. Johnson, majority leader in the U. S. Senate, called for a "politics of unity" which will enable Americans to exert strength and determination in an effort to create a world in which all men can be free. Speaker of the U. S. House of Representatives Sam Rayburn urged greater attention to the task of educating young people in the principles of American government and giving them a desire to perpetuate it.

Total registration through Thursday, with half of the day of the meeting still remaining, had reached 4,727, including 2,742 physicians.

Freedom of Choice

In considering four resolutions which in various ways would have changed or replaced the statements on freedom of choice of physician which the House adopted in June, 1959, when acting upon the recommendations in the report of the Commission on Medical Care Plans, the House reaffirmed the following two statements approved in Atlantic City:

1. "The American Medical Association believes that free choice of physician is the right of every individual and one which he should be free to exercise as he chooses."

2. "Each individual should be accorded the privilege to select and change his physician at will or to select his preferred system of medical care, and the American Medical Association vigorously supports the right of the individual to choose between these alternatives."

However, in order to clarify and strengthen its position on the issue of freedom of choice of physician, the House also adopted this additional statement which was submitted as a substitute amendment on the floor of the House:

3. "Lest there be any misinterpretation, we state unequivocally that the American Medical Association firmly subscribes to freedom of choice of physician and free competition among physicians as being prerequisites to optimal medical care. The benefits of any system which provides medical care must be judged on the degree to which it allows of, or abridges, such freedom of choice and such competition."

Physician-Hospital Relations

The House received 12 resolutions on the

subject of relationships between physicians and hospitals. To resolve any doubt about its position, the House did not act upon any of the resolutions but instead reaffirmed the 1951 "Guides for Conduct of Physicians in Relationships with Institutions." It also declared that "all subsequent or inconsistent actions are considered superceded."

The House also accepted recommendations that (1) the House of Delegates acknowledge the need to strengthen relationships with hospitals by action at state and local levels, (2) the Board of Trustees of the Association continue to maintain liaison with the Board of Trustees of the American Hospital Association, and (3) the Council on Medical Service review this entire problem to ascertain if there have been actions inconsistent with the 1951 Guides.

Those Guides summarize the following general principles as a basis for adjusting controversies:

"1. A physician should not dispose of his professional attainments or services to any hospital, corporation or lay body by whatever name called or however organized under terms or conditions which permit the sale of the services of that physician by such agency for a fee.

"2. Where a hospital is not selling the services of a physician, the financial arrangement if any between the hospital and the physician properly may be placed on any mutually satisfactory basis. This refers to the remuneration of a physician for teaching or research or charitable services or the like. Corporations or other lay bodies properly may provide such services and employ or otherwise engage doctors for those purposes.

"3. The practice of anesthesiology, pathology, physical medicine and radiology are an integral part of the practice of medicine in the same category as the practice of surgery, internal medicine or any other designated field of medicine."

Scholarship Program

To help meet the need for an increasing number of physicians in the future, the House approved the creation of a special study committee which was asked to:

1. Present a scholarship program, its development, administration and the role of the American Medical Association in fulfilling it.

2. Ascertain the maximum to which medical schools could expand their student bodies while maintaining the quality of medical education.

3. Ascertain what universities can support new medical schools with qualified students and sufficient clinical material for teaching—either on a two year or a full four year basis.

4. Investigate the securing of competent medical faculties.

5. Investigate financing of expansion and establishment of medical schools.

6. Investigate financing of medical education as to the most economical methods of obtaining high quality medical training.

7. Develop methods of getting well-qualified students to undertake the study of medicine.

8. Investigate the possibility of relaxing rigid geographic restrictions on the admission of students to medical schools.

The House urged that the special committee be implemented promptly with adequate funds and staff so that it may make an initial report by June, 1960.

Relative Value Studies

Reaffirming a previous policy statement, the House approved in principle the conducting of relative value studies by each state medical society, rather than a nationwide study or a series of regional studies by the AMA. The House also reiterated its authorization for the Committee on Medical Practices to inform each state medical association, through regional or other meetings, of the purpose, scope and objectives of such studies, the steps to be followed in conducting studies, the problems which may be encountered and the manner in which the results can be applied.

The House recognized, however, that some state medical societies are either not interested in relative value studies or are actively opposed to them. It pointed out that some state medical associations fear that the regional conferences of the Committee on Medical Practices will put pressure on them to carry out such studies and that this will result in the adoption of "fixed fees."

Since the regional conferences are educational in nature, the House said, it remains for each state or county medical association to accept or reject the idea of a study in its area.

The House expressed awareness of the fact that this is still a controversial matter. However, it commended the Committee on Medical Practices for its effort to carry out the instructions of the House, and it urged the committee to continue its educational work.

Miscellaneous Actions

In considering 44 resolutions and a large volume of annual, supplementary and special reports, the House also:

Learned that the AMA Board of Trustees has appointed a liaison committee to meet with a similar committee of the *American Osteopathic Association* to consider matters of common concern;

Emphasized that local medical societies should insure that no member violates ethical traditions as they relate to ownership of *pharmacies* or stock in pharmaceutical companies;

Approved the plan of the Committee on Medical Rating of Physical Impairment to publish its new guide on the *cardiovascular system* in the AMA Journal;

Recommended that Association councils and committees, whenever feasible, hold their meetings in the remodeled *Chicago headquarters;*

Called for investigation of the need, desirability and feasibility of establishing a home for *aged and retired physicians;*

Commended *Doctor F. S. Crockett,* retiring chairman of the Council on Rural Health, for his many years of devoted duty;

Urged active promotion and careful study of the newly developed "Guides for Medical Care in *Nursing Homes* and Related Facilities;

Suggested that fees for consultative examinations under programs of the *Bureau of Old Age and Survivors Insurance* should be adjudicated directly between the state medical society and the state agency involved;

Registered a strong protest to the *Veterans Administration,* urging stricter screening of non-service-connected disability patients admitted to government hospitals;

Reiterated the Association's support of the *Blue Shield* concept and directed the Council on Medical Service to submit at the June, 1960, meeting its recommendations concerning a policy statement on AMA relationship with Blue Shield plans;

Suggested that S.J. Res. 41, a bill which would institute a separate program of *international medical research,* be delayed until an over-all assessment can be made of proposals now before Congress dealing with domestic and international medical research;

Endorsed the program of the Educational Council for *Foreign Medical Graduates* but also urged that judicious consideration be given to local problems involved in the July 1, 1960, deadline for certification of foreign graduates;

Urged that *medical schools* include in their curricula a course on the social, political and economic aspects of medicine;

Declared that the threat of *nuclear warfare* has imposed a tremendous responsibility on the medical profession, which must be prepared to assume a critically important role in such an event;

Suggested that the AMA make available to school libraries *information and literature* showing the advantages of private medical care and the American free enterprise system;

Stated that examinations to determine the physical and mental fitness of *aircraft crew members* should be made by doctors of medi-

cine with special knowledge and proficiency in certain techniques;

Urged the American people to get proper *tetanus toxoid,* original and booster, and other immunizations as indicated from their physicians, and called on AMA members to cooperate in an educational program on tetanus immunization;

Recommended that all state and county medical societies establish programs for the inspection and testing of all *fluoroscopes and radiographic equipment*;

Approved the Speaker's proposal that the opening session of the House, at the *Interim Meeting,* be moved from Tuesday morning to Monday morning, with the reference committees meeting on Tuesday and the House reconvening on Wednesday afternoon;

Called upon each individual physician to wage "a vigorous, dynamic and uncompromising fight" against the *Forand type of legislation;*

Urged state and local medical societies and individual physicians to implement the AMA program for recruitment of high-grade *medical students;*

Changed the title of the Section on Surgery, General and Abdominal, to the Section on *General Surgery;*

Accepted with appreciation a $2,500 contribution by Smith, Kline and French Laboratories toward establishment of a suitable award honoring the name of *Doctor Thomas G. Hull,* retiring secretary of the Council on Scientific Assembly, and

Reaffirmed the "Suggested Guides to Relations Between Medical Societies and *Voluntary Health Agencies,*" which were adopted at the December, 1957, meeting in Philadelphia.

At the Tuesday opening session, six state medical societies presented nearly $250,000 to the American Medical Education Foundation. The checks turned over to Doctor George F. Lull, president of AMEF, were: California, $156,562; Indiana, $35,570; New York, $19,546; Utah, $10,355; New Jersey, $10,000, and Arizona, $9,263.

Family Doctors Schedule Philadelphia Meeting

The American Academy of General Practice will hold its 12th Annual Scientific Assembly, March 21-24, 1960, in Philadelphia's Convention Hall. More than 4,000 family doctors and 3,000 residents, interns, exhibitors and wives will attend the four-day program highlighting recent progress in medicine and surgery. The Academy has more than 26,000 family doctor members and is the nation's second largest medical association.

The Philadelphia Assembly program lists 31 prominent medical educators who will discuss a variety of subjects ranging from arthritis and anemia to surgery, geriatrics and mental health. The doctors will also visit more than 100 scientific and 300 technical exhibits. Many of the scientific exhibits will relate directly to the scientific program. They have been carefully selected to answer questions posed by the doctor in active practice. All of the nation's major pharmaceutical firms will be represented in the technical exhibit area.

During the meeting, the doctors will have a chance to be on the other end of a stethoscope during physical examination sessions. More than 600 doctors took physical examinations at the last Academy meeting. Many learned, for the first time, that they needed immediate medical attention.

The scientific program will start at 1:30 p.m., Monday, March 21, and end at noon, Thursday, March 24. The Academy's policy-making Congress of Delegates will meet prior to the scientific sessions, at 2 p.m., Saturday, March 19. All sessions of the Congress and many social functions will be held in the Bellevue-Stratford hotel.

Wednesday evening, March 23, following induction ceremonies for Academy President-elect John Walsh, Sacramento, California, more than 2,000 guests will attend a reception and dance honoring President Fount Richardson, Fayetteville, Arkansas.

The Academy Assembly is this year combined with the Philadelphia Postgraduate Institute.



OSMA President Alfred T. Baker, M.D., Durant, is shown addressing Martin's followers as Doctor Martain, left foreground, and association Vice-President Charles Green, M.D., Lawton, wait their turns.

Doctor Martin Comes Home



Elgin Celebration Attended by 1200

Over twelve hundred grateful friends and patients staged a homecoming celebration on December 6 for Chesley M. Martin, M.D., newly-elected General Practitioner of the Year of the American Medical Association. The event, which took place in the school gymnasium of Doctor Martin's hometown of Elgin, was the climax of nearly a week of personal appearances and television interviews following his selection as the nation's typical family physician.

Mastermind of the hero's welcome was school superintendent Arthur Farrar, Ph.D., who also served as master of ceremonies for the testimonial-type program. Appearing as speakers were: Alfred T. Baker, M.D., Durant, President of the OSMA; Charles Green, M.D., Lawton, OSMA Vice-President; P. A. Becker, Lawton; Maxine Reiff, M.D., Oklahoma City, and William Saupitty, Elgin, leader of the Comanche Tribe.

One of the highlights of the celebration occurred when William Saupitty, Leader of the Comanches, made Doctor Martin an honorary Comanche Chief.



DETAILMEN HONOR DOCTOR MARTIN

The nation's family doctor was doubly honored in Dallas when he received the Medical Service Society of America's Plaque and Key Award from Charles E. Buell, M.S.S.A. President and Dearborn, Michigan representative of Eaton Laboratories.

AAGP Meeting Set For February

The Twelfth Annual Meeting of the Oklahoma AAGP will be held at the Tulsa Hotel, February 15 and 16, 1960. Program Chairman, Marshall O. Hart, M.D., Tulsa, recently announced the completion of an outstanding scientific and social program which is printed below. Further information may be obtained by writing to Doctor Hart, 1228 South Boulder, Tulsa, Oklahoma.

TWELFTH ANNUAL MEETING PROGRAM

Monday, February 15, 1960

8:00 a.m.—Registration

8:55 a.m.—Address of Welcome, Mayor Maxwell, Tulsa

9:00 a.m.—Keith Hammond, M.D., Paoli, Indiana—"Factors Involved in Rational Treatment of U R I in Children"

9:30 a.m.—Benjamin Felson, M.D., Department of Radiology, Cincinnati General Hospital, Cincinnati, Ohio—"Some Fundamentals in Chest Roentgenology"

10:00 a.m.—Visit Exhibitors

11:00 a.m.—James Kelley, M.D., Tulsa, Oklahoma—"Plastic Surgery"

11:30 a.m.—Isadore Dyer, M.D., Tulane University, New Orleans, Louisiana—"Abruptio Placenta, Present Day Concept of Treatment"

12:00 noon—Roundtable Luncheon

1:30 p.m.—Carlo Scuderi, M.D., Chicago, Illinois—"Common Feet Problems"

2:15 p.m.—A. S. Ridolfo, M.D., Indianapolis, Indiana—"Classification and Treatment of Diabetes Whether Orally or Parenterally"

3:00 p.m.—Visit Exhibitors

3:30 p.m.—W. D. Snively, M.D., Medical Director, Mead Johnson, Evansville, Indiana—"Management of Fluid Balance in Obstruction of the Large Bowel"

4:00 p.m.—Benjamin Felson, M.D.—"Some Roentgen Aspects of the Acute Abdomen"

6:30 p.m.—Social Hour

7:30 p.m.—Banquet—Dinner Music by Honey Hudgens (Miss Hospitality of Tulsa)
Speaker will be Fount Richardson, M.D., President of the American Academy of General Practice, Fayetteville, Arkansas—"The Doctor and Politics"
Entertainment by the Student Entertainers, State University, Stillwater, Oklahoma

Tuesday, February 16, 1960

8:00 a.m.—Annual Business Meeting

10:00 a.m.—A. S. Ridolfo, M.D.—"Complication of Diabetes, Including Management During Obstetrical and Surgical Emergencies."

10:30 a.m.—W. D. Snively, M.D.—(Continued) "Management of Fluid Balance in Obstruction of the Large Bowel"

11:00 a.m.—Keith Hammond, M.D.—"Pediatric Errors"

11:30 a.m.—Visit Exhibitors

12:00 noon—Roundtable Luncheon

1:45 p.m.—Carlo Scuderi, M.D.—(Continued) "Common Feet Problems"

2:30 p.m.—Visit Exhibitors

3:30 p.m.—Isadore Dyer, M.D.—"Management of Emergencies in the Third Stage of Labor and Immediate Puerperium"

4:00 p.m.—A. N. Taylor, Ph.D., Associate Dean, University of Oklahoma Medical Center, Oklahoma City, Oklahoma — "Meducation — A 1960 Profile"

4:30 p.m.—Drawing for Major Prize

LADIES' ENTERTAINMENT

Monday, February 15, 1960

Luncheonette, Sky Terrace Room, Tulsa Club

Style Show—Streets Clothiers

AMA, AHA Pledge Health Help for the Needy

The Boards of Trustees of the American Hospital Association and the American Medical Association, in a joint resolution of December 6, announced they will mobilize their full resources to accelerate the development of adequately financed health care programs for needy persons — especially the aged needy.

The resolution made it clear that both organizations will stimulate their state and local components to work with local government toward adequate financing of high quality health care for the needy supported by all community resources and necessary tax funds.

The resolution stated that provision of health care to "the indigent or near indigent is primarily a community responsibility," and made these points:

One: The Boards of Trustees recognize the effective medical care programs for needy persons already established in some states and local communities, but have taken this joint action because of their conviction that such programs deserve increased support and stimulation.

Two: Such proposals as the Forand bill are not designed especially to assist the needy, since they apply to all social security beneficiaries and exclude the majority of needy persons who are not eligible for social security benefits. (Under the Forand bill, sponsored by U. S. Representative Aime Forand [D.-R.I.], some 16 million persons eligible for Social Security benefits—mostly those over 65—would be entitled to receive hospital, surgical and nursing home treatment under a program run by the Federal government. Both the American Hospital Association and the American Medical Association oppose the bill.)

Commenting on the passage of the joint resolution, Doctor Russell A. Nelson, president of the American Hospital Association and director of the Johns Hopkins Hospital, Baltimore, said: "Full payment by states and local communities of the actual costs of care for the indigent and those of low income is still a rarity, although the responsibility for such payment is acknowledged.

"Inadequate payment for high-cost care means that the costs of the care of the needy must be met by the paying patient, by cutting back needed expansion of hospital services, or by payment of insufficient salaries to hospital employees," Doctor Nelson said.

Doctor Louis M. Orr of Orlando, Florida, president of the American Medical Association, said: "The responsibility for taking care of the old person rests primarily with his family. If the family cannot fulfill this responsibility, then it passes to the community, and then to the state.

"We will do everything in our power to encourage local and state governments to fulfill this responsibility.

"Forand-type legislation would reduce the quality of medical care in this country. Such legislation is political medicine."

Text of Resolution

As approved by the Board of Trustees of the American Hospital Association and the Board of Trustees of the American Medical Association, the resolution is as follows:

"WHEREAS The provision of health care to the indigent or near indigent is primarily a community responsibility, and

WHEREAS This responsibility is not being met in many areas because of inadequate financing or poorly planned health care programs, and

WHEREAS Such proposals as HR.4700 (Forand Bill) are not designed especially to assist the needy, since they apply to all Social Security beneficiaries and exclude the majority of needy persons who are not eligible for Social Security benefits, therefore be it

RESOLVED That the Boards of Trustees of the American Hospital Association and the American Medical Association do hereby resolve to mobilize their full resources to accelerate the development of adequately financed health care programs for needy

persons—especially the aged needy—and to stimulate our state and local components to work with local government toward adequate financing of high quality health care for the needy, supported by all available community resources and necessary tax funds. The Boards of Trustees recognize the effective medical care programs for needy persons already established in some states and local communities, but have taken this joint action because of their conviction that these programs deserve increased support and stimulation."

School Health Plan Outlined by Pediatricians

Recommending the employment of private physicians as important members of the school health team, the Committee on School Health of the American Academy of Pediatrics has outlined a health program for school age children.

They advocated the arrangement to be on a part-time basis to bring private physicians into the program further to encourage the doctor's office as the place for detailed examination and treatment.

"Such immunization procedures as are indicated by good pediatric practice should insofar as possible, be done by the patient's private physician or local health department," the Academy committee advised.

Immunizations against diphtheria, tetanus, pertusis, poliomyelitis, smallpox, and others such as typhoid which may be indicated by local conditions, are generally advised by the Committee on Control of Infectious Diseases of the American Academy of Pediatrics, the doctors mentioned, and said that the schools should not take responsibility for performing the actual immunizations.

Describing the function of the school physician, the Academy committee said that he is a health adviser instead of a source of medical care. He is a liaison officer between schools, physicians and health agencies, he advises the school staff on medical matters, he analyzes, with the nurse, the information she has gathered about pupils and their families, and he advises parents and children as to the facilities which exist for solving their medical problems.

"He should not replace or substitute for the child's own physician or other community health service," the pediatricians cautioned.

"The school health team should strive for the cooperation of the local physicians. In turn, the local medical societies and private physicians should cooperate and collaborate with the program," the committee wrote.

They recommended that the school health team employ private physicians part time and give pre-service training courses for them and the entire staff. The team should use standard forms to record examinations and progress, and continuously remind local physicians of the purpose and functions of the school health service. They should make available to private physicians the results of any examinations and appraisals made in the school.

The committee suggested that the medical examination be used as an opportunity to promote preventive medicine, health improvement and health education, rather than solely for the detection of health impairments, and said such a conference should take place in the office of the child's physician.

It is important that health education be integrated into such areas as language, history, science, physical education and social studies, the committee held. Such integration cannot be accomplished unless the teacher has a background of health education or has assistance from the group cooperating in the school health program. It is likewise important that courses in health education be established.

"If the teaching ability of the professional educators can be combined with the medical knowledge and practical experience of the practicing physician and of the public health

physician, children will be better instructed in matters of health and disease," the pediatricians held.

The pediatricians advocated teamwork by private physicians, school physicians, parents, teachers and nurses, and reported:

"The major purpose of a school health program is to maintain, improve and promote the health of the school age child. The program should include adequate supervision of the physical, mental, emotional and social aspects of school life. It also includes planning the course content and instruction in nutrition and health education, including accident prevention, recreation and physical education."

To carry out these objectives, the pediatricians said three services are needed:

1. Health Appraisal. Routine, regular physical examinations, preferably prior to entrance to school, during the intermediate grades, at entrance to secondary school, and before completion of secondary school.

2. Follow-up. It is a waste of time and effort to find the child with a health problem unless something is done about it. Something must be done to insure that the child is placed under adequate medical supervision for the correction of any remediable abnormality found at the time of the examination, or to obtain medical advice as to his adjustment to education in cases where the defect cannot be remedied.

3. Parent Education and Parent Counseling. An important function of the school health program is to educate and counsel the parents in discharging the responsibility that the child's health rests with his parents. It is important to get the co-operation of the parents and to motivate them to heed their physician's advice in obtaining the necessary medical care.

The pediatricians outlined details of the parts each member of the team plays. Parents should be alert to notice changes in their children's appearance or actions which suggest need for medical advice, they counseled.

Teachers may be prepared through their interests and education to observe pupils for deviations from normal appearance and behavior.

"Not all teachers are adequately prepared or equipped to assume this responsibility," the report said. "Some teachers are not good observers. Their many pedagogic duties are naturally their major interest. In-service training for their part in the school health program is essential."

The committee recommended that the teacher transmit her impressions to the physician or school nurse and to the parents only if there is no intermediate medical personnel in the school. "If she consults directly with the parent, she should be very careful not to make a diagnosis, which, if wrong, could be damaging to both the parents and child. Instead, she should point out apparent functional deviations and recommend medical attention. Her exact procedure will depend on the individual case, but will always call for tact and discrimination."

The committee praises the importance of the teacher, and that of the school nurse. The nurse is at the center of activities and has direct relationships with teachers, physicians, health officials and parents. The nurse helps train teachers in observation and screening. She assists the school physician, prepares records and medical histories, and is a counselor and friend to the parent. Her position in relation to the community was emphasized:

"She relates the health services in schools to the work of other community health services, and many times she is the interpreter or coordinator of health activities within the school. She has an important role in interpreting handicapped children to the school staff, and she works with guidance, psychologic and special education services on their behalf.

"Much of her work can be accomplished through the school health council; on it the nurse is usually the motivating and coordinating force."



New members of the Tulsa County Medical Society honored at the annual indoctrination dinner on December 8th at Tulsa are: Left to right, seated—Kenneth L. Duncan, M.D.; C. Frank Knox, Jr., M.D.; James W. Kelley, M.D., Society President; David C. Clemans, M.D., Skiatook; Robert L. Lubin, M.D., Chairman of the Medical Precepts Committee; Francis W. Pruitt, M.D., and Dwane B. Minor, M.D. Standing, left to right—Nathan A. Cohen, M.D.; Richard A. Liebendorfer, M.D.; Kenneth L. Knuth, M.D.; John E. Kauth, M.D.; William L. Harris, M.D.; Joe Burge, M.D.; and Jack E. Hale, M.D. Not present when photograph was made were Jack W. Newport, M.D.; Hobart C. Sanders, M.D., and Duane E. Brothers, M.D.

Tulsa County Society Welcomes New Members

Fifteen new doctors who entered practice in Tulsa County during 1959 were formally welcomed into the Tulsa County Medical Society last month.

The occasion was the Society's Annual Indoctrination Dinner for New Members held at the Mayo Hotel on Tuesday, December 8, 1959.

The newcomers heard lectures on medical ethics, local customs of practice, malpractice prevention and defense, current federal medical legislation, the doctor and the press, and financial aspects of private practice.

Guest speakers included Mr. Richard M. Nelson, Field Representative for the American Medical Association, Chicago, Illinois; Mr. Truman B. Rucker, Tulsa attorney and legal counsel for the Tulsa County Medical Society; Walter F. Sethney, M.D., Chairman of the Ethics Committee; John E. McDonald, M.D., Chairman of the Legislative Committee; Marshall O. Hart, M.D., Chairman of the Medical Advisory Committee; James W. Kelley, M.D., President; and Mr. Jack Spears, Executive Secretary.

The program, an annual event first begun in 1954, was under the direction of the Society's Medical Precepts Committee. Robert I. Lubin, M.D., Chairman, presided.

New doctors honored at the dinner included:

Joe Burge, M.D., thoracic surgeon; Duane E. Brothers, M.D., anesthesiologist; David C. Clemans, M.D., general practitioner, Skiatook; Nathan A. Cohen, M.D., pathologist; Kenneth C. Duncan, M.D., neurologist; Jack E. Hale, M.D., pathologist; William L. Harris, M.D., general practitioner; John E. Kauth, M.D., radiologist; Kenneth L. Knuth, M.D., radiologist; C. Frank Knox, Jr., M.D., psychiatrist;

Richard A. Liebendorfer, M.D., internal medicine; Dwane B. Minor, M.D., dermatologist; Jack W. Newport, M.D., orthopedic surgeon; Hobart C. Sanders, M.D., gynecologist and obstetrician; and Francis W. Pruitt, M.D., director of postgraduate education at Hillcrest Medical Center.

wherever there is inflammation, swelling, pain

VARIDASE®

Streptokinase-Streptodornase Lederle

BUCCAL Tablets

conditions for a fast & comfortable comeback

Host reaction to injury or local infection has a catabolic and an anabolic phase. The body responds with inflammation, swelling and pain. In time, the process is reversed. VARIDASE speeds up this normal process of recovery. By activating fibrinolytic factors VARIDASE shortens the *undesirable phase,* limits necrotic changes due to inflammatory infiltration, and initiates the constructive phase to speed total remission. Medication and body defenses can readily penetrate to the affected site; local tissue is prepared for faster regrowth of cells. In infection, the fibrin wall is breached while the infection-limiting effect is retained. In acute cases, response is often dramatic. In chronic cases, VARIDASE Buccal Tablets can stimulate a successful response to primary therapy previously considered inadequate or failing.

for routine use in injury and infection ...new simple buccal route

VARIDASE Buccal Tablets should be retained in the buccal pouch until dissolved. For maximum absorption, patient should delay swallowing saliva.
Dosage: One tablet four times daily usually for five days. When infection is present, VARIDASE Buccal Tablets should be given in conjunction with ACHROMYCIN® V Tetracycline with Citric Acid.
Each VARIDASE Buccal Tablet contains: 10,000 Units Streptokinase and 2,500 Units Streptodornase.
Supplied: boxes of 24 and 100 tablets.

1. Innerfield, I.: Clinical report cited with permission
2. Clinical report cited with permission

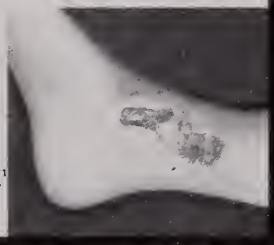
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Pearl River, New York





FORCE INJURY
severe bruises ... swelling ... cleared by fifth day[2]





VARICOSE ULCER
15 years duration ... resolved with VARIDASE[1]





INFLAMMATORY DERMATOSIS
rapidly spreading rhus dermatitis healed within a week[1]





INFECTED LACERATION
marked reversal in 3 days... returned to school... closure advanced[1]





THROMBOPHLEBITIS
back on his feet in a week after recurrent episode[1]





REFRACTORY CELLULITIS
normal routine resumed after 4 days of VARIDASE[1]

Health Problems Discussed on New Radio Series



Script for a new radio series concerning family health problems are discussed by Doctor Gerald Dorman (right) of the American Medical Association, Doctor Benjamin W. Carey (left), of Lederle Laboratories, and American Broadcasting Company's John Daly. The three organizations are cooperating to present the public service series, called "Highroad to Health." It is being carried nationally by the ABC radio network. Each program in the weekly series presents in dramatic form a health problem commonly encountered by American families, followed by a discussion of the problem by a distinguished guest physician. Doctor Dorman is Co-Chairman of the Physcian's Advisory Committee on Radio and TV of the AMA. Doctor Carey is medical director of American Cyanamid Company's Lederle Laboratories and Mr. Daly, television and radio personality, ABC vice president for public affairs. Local program listings or ABC stations should be consulted for exact broadcast schedules for "Highroad to Health."

PARTICIPATE IN YOUR ANNUAL MEETING!

HOW? By preparing a SCIENTIFIC EXHIBIT or by documenting a BIZARRE CASE as described on pages 52, 53.

WHEN? Applications must be received prior to March 1, 1960. The meeting will be held in Oklahoma City on May 2, 3 and 4.

WHY? It will be the best meeting ever . . . a real opportunity to share your experiences with over half of the OSMA membership.

Texas University Announces Medical Cruise

The University of Texas Postgraduate School of Medicine has announced plans for the Annual Texas Medical Cruise to the Caribbean, May 6, to May 19th, 1960.

Faculty for the fourteen day cruise will be Alfred E. Leiser, M.D., Clinical Instructor in Medicine, Baylor University College of Medicine and Clinical Assistant Professor of Medicine, University of Texas Postgraduate School of Medicine, Houston, Texas; L. Maxwell Lockie, M.D., Professor and Head of the Department of Therapeutics, University of Buffalo School of Medicine, Buffalo, New York; Jarrell E. Miller, M.D., Clinical Professor of Radiology, University of Texas Southwestern Medical School, Dallas; and Fred M. Taylor, M.D., Associate Professor of Pediatrics, Baylor University College of Medicine, Houston, Texas.

The cruise will depart from New Orleans on May 6 and return to New Orleans on the 19th. Ports of call will include: Havana, Cap Hatien, Port-Au-Prince, Montego Bay and Grand Cayman.

The itinerary will allow 25 hours of Category I teaching credit for physicians desiring AAGP credit.

OB-GYN Examinations

Examinations (Part II), oral and clinical will be held in Chicago at the Edgewater Beach Hotel from May 11 through 16, 1960. Details available from Robert L. Faulkner, M.D., Secretary, 2105 Adelbert Road, Cleveland 6, Ohio.

REQUEST FOR JOURNALS

The Journal of the Oklahoma State Medical Association would appreciate receiving copies of the 1959 June and September issues of THE JOURNAL. Please mail to P.O. Box 9696, Oklahoma City, Oklahoma.

Have You Heard?

PAT FITE, SR., M.D., Muskogee, attended the Western Surgical Meeting in Colorado Springs during November.

RALPH A. MCGILL, M.D., Tulsa surgeon, was recently presented a plaque recognizing his twenty years' service as member and chairman of the Hillcrest Medical Center's board of governors.

FRANCIS E. SMITH, M.D., formerly of Kingfisher, has recently opened a new clinic in Norman, located at Comanche and Lahoma streets.

R. L. CURRIE, M.D., Stilwell, and R. H. MAYES, M.D., Duncan, have been appointed County Superintendents of Health of their respective counties, Adair and Jefferson.

ROBERT C. LAWSON, M.D., Oklahoma City, has been appointed to the Board of State Governors of the American Diabetes Association. Purpose of the Board is to stimulate interest in diabetes and related conditions, as well as facilitate coordination of activities in this field.

J. B. HOLLIS, M.D., Mangum, has been appointed Chairman of the National Rehabilitation Committee of the American Legion. Doctor Hollis, who has served in various capacities in the Legion for over forty years, will be in charge of the benefit program on a national scale.

WILLIAM L. BYNUM, M.D., has opened offices in the Barnes Building in Muskogee where he will specialize in Obstetrics and Gynecology. A graduate from the University of Arkansas Medical School, Doctor Bynum has completed a year's residency at the Arkansas Medical Center in Little Rock.



Have something to exhibit

If so, you should apply now for space at the 1960 meeting of your association. Scientific Exhibits will again play a major role . . . so why not prepare a graphic presentation of your work and share it with the 700 physicians who will attend the state's largest medical meeting? A research or clinical project may be shown as an individual exhibit, or you may present a single case in the "Klinical Kaleidoscope" section.

Research or clinical project

Perhaps you have done some original research, undertaken some experimental work, or simply drawn some conclusions based upon a series of interesting cases. Then you would be eligible to apply for an exhibit booth of your own in the Scientific Exhibit Section. A subject which has a special visual interest would be particularly suitable . . . and it need not be large—just stimulating!

Bizarre case

All of us have encountered a unique case which is worth talking about. If you have a well-documented one which you would like to share with others, toss your hat in the ring today by completing the application on the opposite page. The "Klinical Kaleidoscope" section will be a varied selection of bizarre cases, each presented with one to five x-rays or illustrations and accompanied by a very brief abstract of the clinical course.

O.S.M.A. ANNUAL MEETING

municipal auditorium · oklahoma city

may 2·3·4, 1960

APPLICATION FOR SCIENTIFIC EXHIBIT SPACE

54th Annual Meeting

OKLAHOMA STATE MEDICAL ASSOCIATION

Municipal Auditorium — Oklahoma City — May 2, 3, 4, 1960

1. NAME AND ADDRESS OF APPLICANT:

2. TITLE OF EXHIBIT:

3. INDIVIDUAL BOOTH? ☐ "KLINICAL KALEIDOSCOPE" SECTION? ☐

4. SPACE REQUIRED:

   A. BOOTH: Indicate width in multiples of eight feet _____

   (All booths will be 6 feet in depth)

   B. "KLINICAL KALEIDOSCOPE": Indicate square footage:

   Wall-type display _____ Table top _____

5. A. GENERAL DESCRIPTION:

   B. MATERIAL TO BE SHOWN (Subject matter):

   C. METHOD OF PRESENTATION (x-ray, photos, graphs, models, etc.):

6. PREVIOUS SHOWINGS

ACT NOW — SPACE IS LIMITED

Applications Must Be Received Prior to March 1, 1960

Oklahoma State Medical Association • Box 9696 • Oklahoma City

Auxiliary News

Safe New Year

What more appropriate way can be found to start the new year than by personal participation in making our world a safer place in which to live? Startling daily statistics in our public and private lives keep us constantly aware of the need to do "something." Our National Safety Chairman interprets our Safety Program as being three dimensional: you, your family, and your community.

Accident Prone?

Obviously, Auxiliary members can begin with evaluation of home safety. The phrase "accident-prone," which has been over-used during the last few years might in many cases give us cause for pause. If we apply the description rather loosely to one of our own children or to a family member, can we objectively say that our own negligence or carelessness is never contributory?

Opportunity Unlimited

Safety Chairmen perform one of our most important functions. Her realm is unlimited. Cooperation of members must be constructive on a community basis. After evaluating your own town or city, the Safety Committee can present its findings to your Auxiliary for specific action. In few fields can so much be accomplished. Include, in addition to specific home safety improvements, your schools, your churches, your public buildings, your traffic problems. Achieve some good public relations, too, by working with various groups in your city. Find interested allies in your local Safety Council, Civil Defense, your city or county Safety Commissioners.

In our effort to combat legislation and problems of aging, make a survey of Safety angles involved. Are rest homes in your area safe? Are county home facilities adequate from a safety angle? Do your Golden Age Clubs have material and instruction for driving and pedestrian safety?

Safety Goals—Practical Insurance

Our goals include educating each car-owner to the value of installing seat belts—and using them; educating our buying public to secure safety features as standard equipment for all cars; and by encouraging family car-owners to install safety features designed for the protection of their children. (Especially important is the follow-through on the last suggestion: teach small children good "car manners" as an intelligent approach to discipline and safety.)

Highway safety can include safe driver education programs for all drivers; periodic re-examination for drivers' licenses; education on chemical tests for intoxication; and factors in pedestrian safety and bicycle safety instruction.

Greater home and farm safety presents an inexhaustible array of material; particularly stressed is the establishment of poison control centers. (Every year over 1,000 Americans, largely children, die of accidental poisoning.)

We hope that as you have been reading, you have already focused a challenging eye upon some subject very close to yourself and your community. Your "individual responsibility" can be its effective best in promoting Safety to the utmost. If you need help in any way, your State Safety Chairman, Mrs. Floyd Bartheld, 530 East Creek, McAlester, is waiting to hear from you.

We wish for you a Safe New Year!

What's Your Hobby, Doctor?

The DOCTOR'S HOBBY SHOW has become one of the outstanding attractions at the OSMA ANNUAL MEETING. A project of the Woman's Auxiliary, the show offers physicians an excellent opportunity to display the products of their leisure time. If you have a hobby, don't keep it a secret . . . Show your colleagues what you can do . . . APPLY NOW!

Doctor's Hobby Show

O.S.M.A. Annual Meeting

Municipal Auditorium

Oklahoma City

MAY 2, 3, 4, 1960

Application For Hobby Show Space

54th ANNUAL MEETING

OKLAHOMA STATE MEDICAL ASSOCIATION

DESCRIBE EXHIBIT, including information as to size, shape and value (insurance is provided):

IMPORTANT: Deliver Exhibit to Zebra Room, Municipal Auditorium by noon, May 1. Your Exhibit will be personally attended and insured at all times. It must be picked up by noon, May 4, when management responsibility ends.

MAIL THIS FORM TODAY!

State Chairman—Mrs. William R. R. Loney
2440 E. 26th Place
Tulsa 14, Oklahoma

State Co-Chairman—Mrs. Harrell C. Dodson, Jr.
1108 N.E. 42nd Street
Oklahoma City, Oklahoma

Army Needs Doctors

The Surgeon General of the United States Army has announced the need for Doctors of Medicine for assignment to U. S. Army Medical facilities throughout the world.

Doctors who specialize in all branches of the Medical profession, with the exception of Pediatricians, Anesthesiologists, and Ophthalmologists are encouraged to apply for active duty with the U. S. Army. Applicants, upon acceptance, will be called to active duty for three years or they may volunteer for indefinite tours beginning in April of 1960.

Grades of appointees will be determined by number of years of participation in professional activities. Medical school and internship will be counted both for pay and for determining initial grade of applicant.

For further details and applications, interested Doctors in Arkansas, Louisiana and Oklahoma should contact Lt. Col. W. H. Dickerson, Headquarters, Oklahoma Sector Command, XIX U. S. Army Corps (Reserve), 1101 N. Broadway, Oklahoma City, Oklahoma, Telephone: CEntral 5-4552.

College of Surgeons to Hold Colorado Meeting

Surgeons and related medical personnel are invited to attend the three-day Sectional Meeting of the American College of Surgeons in Colorado Springs, Colorado, March 21 through 23, 1960. Headquarters will be The Broadmoor Hotel.

The program will include scientific reports on topics of current concern such as management of the injured hand, treatment of duodenal ulcer, appendicitis, treatment of neck fractures, surgical convalescence, and cancer and pregnancy. Symposiums on injuries, cancer and hospital infections will be presented. Also, selected medical motion pictures and cine clinic films from the 1959 Clinical Congress will be shown.

Wednesday, March 23, Doctor Paul R. Hawley, Chicago, The Director, American College of Surgeons, will be the speaker at The College dinner. The subject of his talk will be, "The Epidemiology of An American Legend."

Coming Meetings

UNIVERSITY OF OKLAHOMA MEDICAL CENTER
POSTGRADUATE PROGRAM*
Individual Postgraduate Courses

BASIC ELECTROCARDIOGRAPHY — February 29 through March 5.

OPTHALMOLOGY-OTOLARYNGOLOGY SYMPOSIUM—March 3 and 4.

OBSTETRICS-GYNECOLOGY SYMPOSIUM—March 5.

ADRENAL STEROIDS—March 24, 25, and 26. (Third Oklahoma Colloquy on advances in Medicine.)

ORTHOPEDIC SYMPOSIUM—May 13 and 14.

CARCINOMA OF THE SKIN—April 22 and 23. (Sixth Annual Combined Surgery, Radiology, Pathology Symposium.)

OKLAHOMA ASSSOCIATION OF HOUSE STAFF PHYSICIANS—May 6.

PEDIATRIC NEUROLOGY—May 12 and 13. This symposium to be held in conjunction with the Wednesday Short Course on May 11.

SERIAL POSTGRADUATE COURSE

Postgraduate Division

Oklahoma City, Oklahoma
1959-1960

Wednesday Short Courses
3:30 to 8:30 p.m.

Jan. 13	Carl Puckett Memorial Lecture and Infectious Diseases Symposium Guest Lecturer: William Tucker, M.D., Washington, D.C.
Feb. 10	Rehabilitation of the Cardiac Patient
Mar. 9	C. B. Taylor Lectureship and Urology Symposium—Guest Lecturer to be announced
Apr. 13	Anesthesia for the Part-time Anesthetist
May 11	Neurological Diseases in Childhood
June 8	Surgery

*Further information may be obtained from the Office of Postgraduate Education, University of Oklahoma School of Medicine, 801 N.E. 13th Street, Oklahoma City, Oklahoma.

OKLAHOMA CHAPTER
AMERICAN ACADEMY OF GENERAL PRACTICE
February 15-16, 1960 — **Tulsa Hotel**
Tulsa, Oklahoma

The 12th Annual Meeting of the Oklahoma Chapter of the American Academy of General Practice will be held at the Tulsa Hotel, February 15-16, 1960, in Tulsa. Complete program on page 43.

SCOTT AND WHITE CLINIC
EIGHTH ANNUAL POSTGRADUATE CONFERENCE
March 6, 7, 8, 1960 — **Temple, Texas**

This course, Current Topics in Medicine and Surgery, will be presented by Scott, Sherwood and Brindley Foundation and the University of Texas Postgraduate School of Medicine on March 6, 7, 8, 1960. Registration fee is $25.00 and the course is approved for 20 hours credit by AAPG. Further information is available by writing to Publications Office, Scott and White Clinic, Temple, Texas.

EIGHTH POSTGRADUATE COURSE
IN
DIABETES AND BASIC METABOLIC PROBLEMS
January 20, 21, 22, 1960 — **Ambassador Hotel**
Los Angeles, California

The Eighth Postgraduate Course in Diabetes and Basic Metabolic Problems will be offered by the American Diabetes Association, January 20, 21, 22, 1960 at the Ambassador Hotel in Los Angeles. The course is accepted for 18 hours credit, Category II, by the American Academy of General Practice. Detailed information is available by writing to American Diabetes Association, Inc., 1 East 45th Street, New York 17, New York.

AMERICAN COLLEGE OF ALLERGISTS
Graduate Instructional Course and Annual Congress
February 28-March 4, 1960 — **The Americana Hotel**
Bal Harbor
Miami Beach, Florida

The American College of Allergists will hold a Graduate Instructional Course and Annual Congress February 28 to March 4, 1960 at the Americana Hotel, Bal Harbor, Miami Beach, Florida. For further information, contact John D. Gillaspie, M.D., Treasurer, 2049 Broadway, Boulder, Colorado.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY
March 7-10, 1960 — **Roosevelt Hotel**
New Orleans, Louisiana

The twenty-third annual meeting of The New Orleans Graduate Medical Assembly will be held March 7-10, 1960 in New Orleans. Complete details may be obtained by writing to Mannie D. Paine, Jr., M.D., Secretary, The New Orleans Graduate Medical Assembly, 1430 Tulane Avenue, New Orleans 12, Louisiana.

Deaths

Shade Durrett Neely, M.D.
1897-1959

Shade Durrett Neely, M.D., a practicing physician in Muskogee since 1922, died at his home November 19, 1959.

Born in Charleston, Mississippi on July 1, 1897, Doctor Neely graduated from the University of Oklahoma School of Medicine in 1920. After postgraduate work in Baltimore and New York City he established his practice in Muskogee where he specialized in urology.

Active in the Medical Profession for many years, Doctor Neely was a past president of the East Central Oklahoma Medical Society and at one time served as Councilor from District IX of the Oklahoma State Medical Association.

In addition to his work in the medical profession, he was active in the Masonic Lodge, having served in various capacities in Oklahoma and was an honorary member of the Masonic orders in Kansas, Texas, Arkansas, Indiana and Mississippi.

Samuel C. Rutherford, M.D.
1869-1959

Samuel C. Rutherford, M.D., 90-year-old Locust Grove physician, died in Pryor, November 13, 1959.

Born in 1869 at West Fork, Arkansas, Doctor Rutherford graduated from the University of Arkansas School of Medicine in 1901. Practicing first in Chouteau, Indian Territory, then at Wagoner, he came to Locust Grove in 1924 where he had practiced since.

Doctor Rutherford was honored by the Oklahoma State Medical Association in 1950 when he was presented with a Life Membership in recognition of his years of service to the medical profession.

Henry A. Ellis, M.D.
1876-1959

Henry A. Ellis, M.D., 84-year-old Kiowa physician, died November 13, 1959 in McAlester.

A native of Lidell, Mississippi, Doctor Ellis graduated from the College of Physicians and Surgeons in Dallas in 1905. After practicing in Mississippi for a short while, he came to the Chickasaw nation in Indian Territory, settling near Tishomingo, later moving to a community near Coalgate. Since 1914, he has maintained offices in Savanah, Kiowa and Pittsburg.

In 1952, Doctor Ellis was honored for his years of service to the medical profession when the Oklahoma State Medical Association presented him with a Life Membership.

25 YEARS AGO



Articles published in *The Journal* of the Oklahoma State Medical Association, January, 1935.

EDITORIAL
PUBLIC POLICY AND LEGISLATION IN 1935

There never has been a time since statehood when organized medicine in Oklahoma was in a more advantageous position to accomplish great good for the people in the state than this year, 1935.

We have come through the past four years with our "heads up" and our position well defined, notwithstanding the most serious opposition of the administration, this including the Board of Regents of the University. Even the Medical School has had to suffer from petty politics in dividing the administration of the University Hospital, thereby creating a spirit of unrest and uncertainty from the staff to the janitor, but the members of the medical profession connected with the school have stood fast and brought the institution through with no loss in its scholastic classification and the University Hospital still maintains its standardization. Post-graduate medical teaching has been carried on by the State Medical Association after the Board of Regents passed a resolution opening these courses to the cults, making it impossible for organized medicine to go on with this important phase of medical education with the aid of the University.

So much for our troubles and accomplishments during the past four years. Now let us look forward to the possibilities of 1935, under a state administration headed by a man of sound reasoning, who has indicated that he will favor any measure that will protect the people of this state and forward an intelligent program in the prevention and treatment of disease.

The legislative committee of this association has been active for several months; they have given careful consideration to many proposed bills affecting the medical profession and the health and safety of our people.

The crippled children's law must be changed to meet present conditions; a basic science law will do much to eliminate the unprepared candidate to practice the healing art in any of its branches; the rescinding of the resolution by the Board of Regents opening post-graduate medical teaching to the cults; the appointment of a doctor of medicine on the Board of Regents of the University; the protection of doctors and hospital fees in cases of automobile accidents, and many other items of legislation and policy will be under consideration.

Now comes the important point of this editorial—every member of the Oklahoma State Medical Association must accept his individual responsibility and use his influence to bring about a proper solution of these problems. If each physician who does the family practice for a member of the legislature would let him know his attitude toward these important questions there would be no doubt as to the accomplishment of this entire program. This is very little to expect from the individual doctor and as the legislature will be in session when you read this, let me suggest that you contact your senator or representative at once and let us take advantage of an intelligent administration to have enacted a set of intelligent laws, thereby promoting the health and happiness of the people for whom we are responsible.

NEWS OF THE COUNTY MEDICAL SOCIETIES

ALFALFA-WOODS County Medical Societies held their annual meeting for election of officers for 1935 and the following were elected:

Alfalfa County

President—J. P. Bishop, Aline.
Vice-President—Z. J. Clark, Cherokee.
Secretary—L. T. Lancaster, Cherokee.

Woods County

President—I. F. Stephenson, Alva.
Vice-President—C. A. Tracerse, Alva.
Secretary—O. E. Templin, Alva (re-elected).
Delegate—D. B. Ensor, Hopeton.

Thirty six crippled children were examined at the meeting by Doctors John Day, Supply; W. K. West and Harry Wilkins both of Oklahoma City.

Book Review

COLD INJURY, GROUND TYPE, Office of the Surgeon General, Department of the Army, U. S. Printing Office, Washington, D.C., 1958, pp. 570.

In the Southwest cold injury is uncommon. This volume is, to the reviewer's knowledge, the most complete treatise available on this subject. It is of historical and clinical importance, and should be available for all physicians engaged in general medical and surgical practice. Tissue damage due to cold at altitude, at ground level, in the tropics, and that associated with moisture with their pathogenesis, diagnosis, treatment, and epidemiology are described in separate sections. Care of the feet and their protection is emphasized. In the event of a mass disaster in the winter, the lessons learned and described can be beneficially applied to civilian populations and individuals.—*John A. Schilling, M.D.*

PHYSICIAN PLACEMENT

Dermatology

Thomas E. Slimp, M.D., 323 West Livingston Place, Metairie, Louisiana, age 42, married, Syracuse Medical School, 1950, board certified January 1, 1960. No military obligations, available January 1, 1960.

Gordon Harold Ekblad, M.D., Field Medical Service School, Camp Pendleton, California, 53, married, University of Minnesota, 1930, available January 1960.

General Practice

Johnny Bill Delashaw, U.S.P.H.S. Hospital, Wyman Park Drive and 31st Street, Baltimore 11, Maryland, age 27, married, University of Texas Medical Branch, 1959, available July 1960.

D. H. Bessesen, M.D., Grand Canyon Hospital, Grand Canyon, Arizona, age 61, married, University of Minnesota, interested University Health Service, available October 1, 1959.

Sherman Allen Hope, M.D., 1105 Yale Ave., Panama City, Florida, PO 3-6656, age 27, married, University of Oklahoma School of Medicine, 1957, available July 1960.

Paul J. Vega, M.D., 6070 Orleans Ave., New Orleans, Louisiana, age 26, married, Louisiana State University, 1956, no military obligations, available now.

Thomas S. Whitecloud, M.D., 858 Market Street, Pascagoula, Mississippi, age 45, married, Tulane, 1943, veteran, has interest in teaching, availability date depends on situation.

Internal Medicine

Richard I. Hochman, M.D., 37, Badger Road, Annapolis, Maryland, 31, married, New York University, 1952, available October 1, 1960.

Albert D. Roberts, Jr., M.D., 4333 Colgate, Dallas, Texas, 29, married, Southwestern Medical School, Veteran, available July 1959.

Van B. Saye, Jr., M.D., 1230 Brockenbraugh Court, Metairie, Louisiana, age 28, married, Medical College of Georgia, 1954, Air Force Reserve, available July 1, 1960.

Locum Tenens

Harold W. Calhoon, M.D., Wesley Hospital, Oklahoma City. One year residency in Internal Medicine. Available July 1 to September 15th, 1960.

Arnold Giesbrecht, M.D., Hallock, Minnesota, age 34, married, graduate Winnipeg, Manitoba, 1957, prefers suburban or locality close to cities.

James D. Green, M.D., second year resident in Internal Medicine at St. John's Tulsa, desires one year of Internal Medicine practice before taking third year of training. Prefers clinic in city of 50,000 or greater population.

Russell M. Preston, M.D., 476 San Juan, Oak Harbor, Washington, desires six month's work in General Practice between time of separation from military service to beginning of residency training, January-July, 1960.

Obstetrics and Gynecology

Lynn W. Abshere, M.D., 708 N.E. 14th, Oklahoma City, age 31, married, University of Oklahoma School of Medicine, 1956, American College of OB-GYN, no military obligations, available July 1, 1960.

Gerald R. Keilson, M.D., Medical Arts Building, Dallas, Texas, age 31, married, University of Texas, 1953, board qualified, veteran, available in July 1960.

Milton Gardner Mutch, Jr., M.D., 423 Mills Avenue, Fargo, North Dakota, age 31, married, University of Minnesota, 1955, board eligible upon completion of fellowship, veteran, available July 1, 1959.

David H. Holmes, M.D., 1017 Grovena Drive, St. Louis, Missouri, 37, married, Washington University, St. Louis, Veteran, available July 1, 1960.

Nejdat Mulla, M.D., 1110 Belmont Avenue, Youngstown, Ohio, age 36, married, University of Geneva, Switzerland, 1952, American College of OB-GYN, not eligible military service, available July 1, 1960.

Harold G. Ray, M.D., 212 Darien Place, Box 503, Balboa Heights, Canal Zone, age 28, married, University Arkansas School of Medicine, 1955, now in active reserve, available July 1, 1960.

Alvon C. Winegar, M.D., 1940 Michigan, Benton Harbor, Michigan, age 41, married, Wayne University, 1942, no military obligation, available early 1960.

Pediatrics

H. M. McClintock, M.D., 5712 St. John St., Kansas City, Missouri, 29, married, Baylor University, 1955, veteran, available December 1960.

Surgery

(Name on Request) 32 years old, married, Tulane, 1952, veteran, board eligible.

Johnson P. Graves, M.D., Box 142, Pulaski Heights Station, Little Rock, Arkansas, age 36, married, Arkansas School of Medicine, 1950, board eligible, no military obligations, available Fall 1959.

Olin O. Williams, Jr., M.D., 3768 Alma Drive, Memphis, Tennessee, 29, married, University of Tennessee, veteran, available January 1962.

Urology

Charles Lee Reynolds, Jr., M.D., 5012 Rockport Drive, Dallas, Texas, age 32, Southwestern Medical College, Dallas, 1949, board eligible, married, no military obligations, available July 15, 1960.

Alex Grossman, M.D., V.A. Center, Temple, Texas, 42, married, University of Texas, 1951, board certified, veteran, available now.

Herbert Sohn, M.D., 2010 Cliffview Road, Cleveland, Ohio, 32, married, Chicago Medical School, 1955, veteran, available July 1960.

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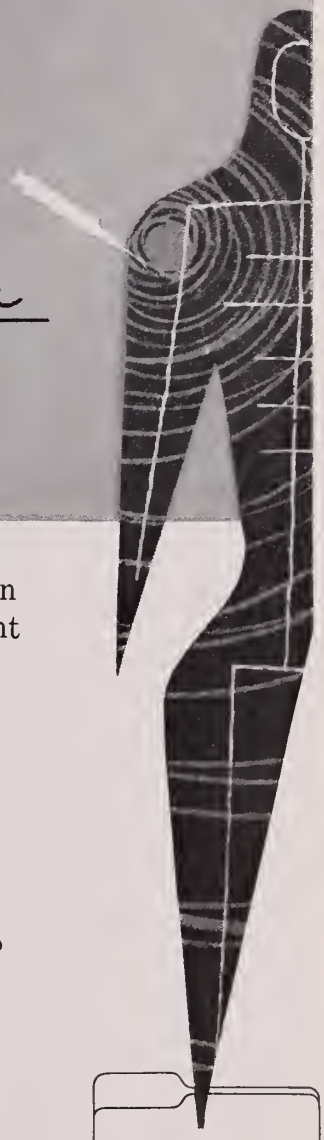
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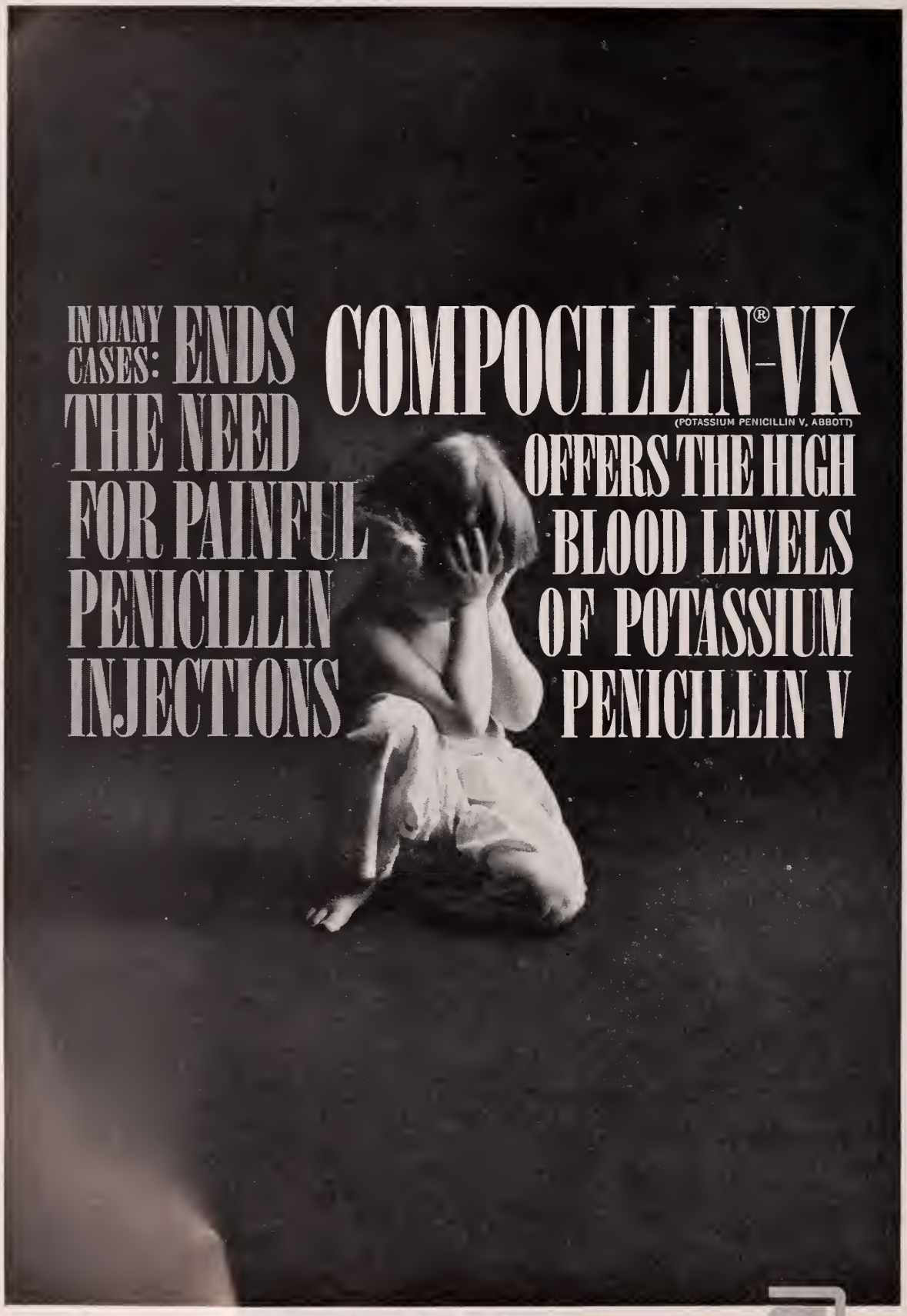
*Thompson, R. E., and Hecht, R. A.: Am. J. Clin. Nutrition 7:311-317 (May-June) 1959.

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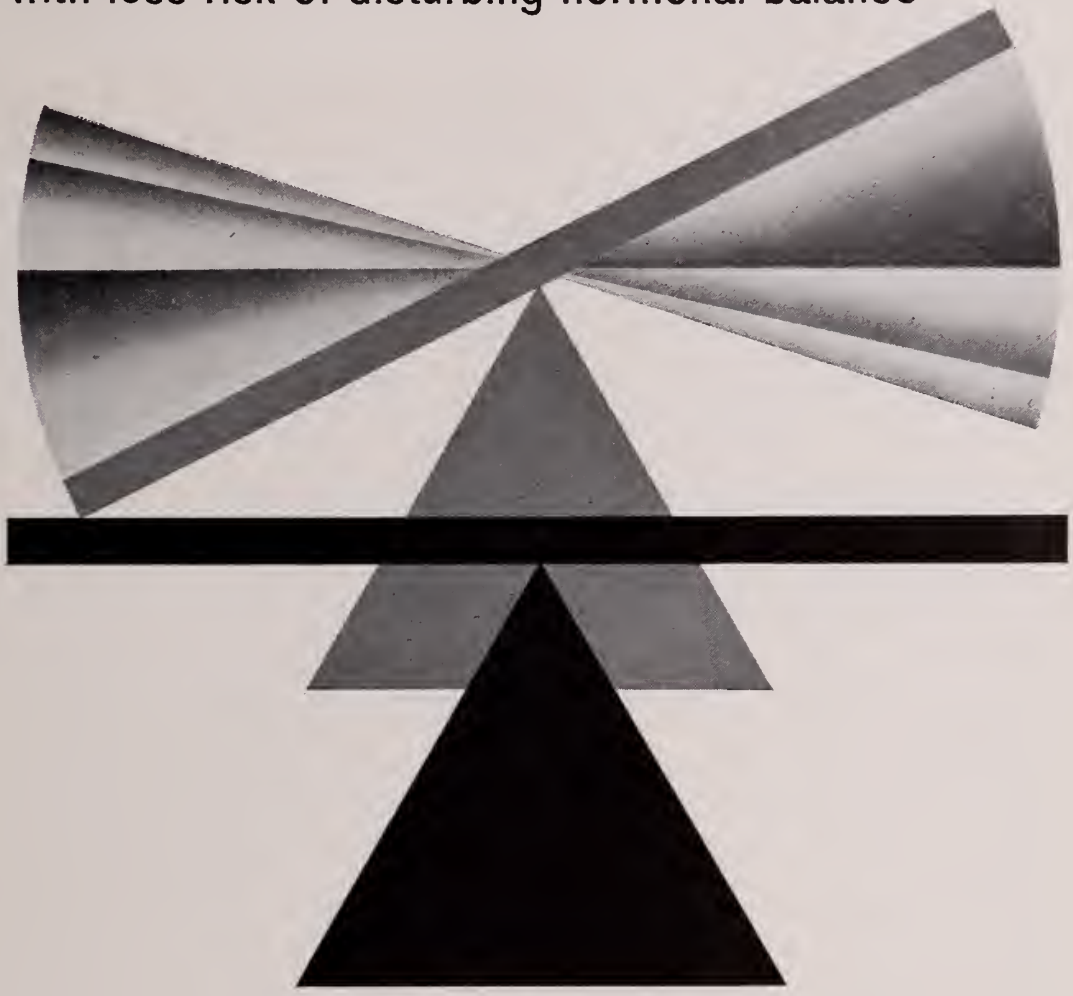
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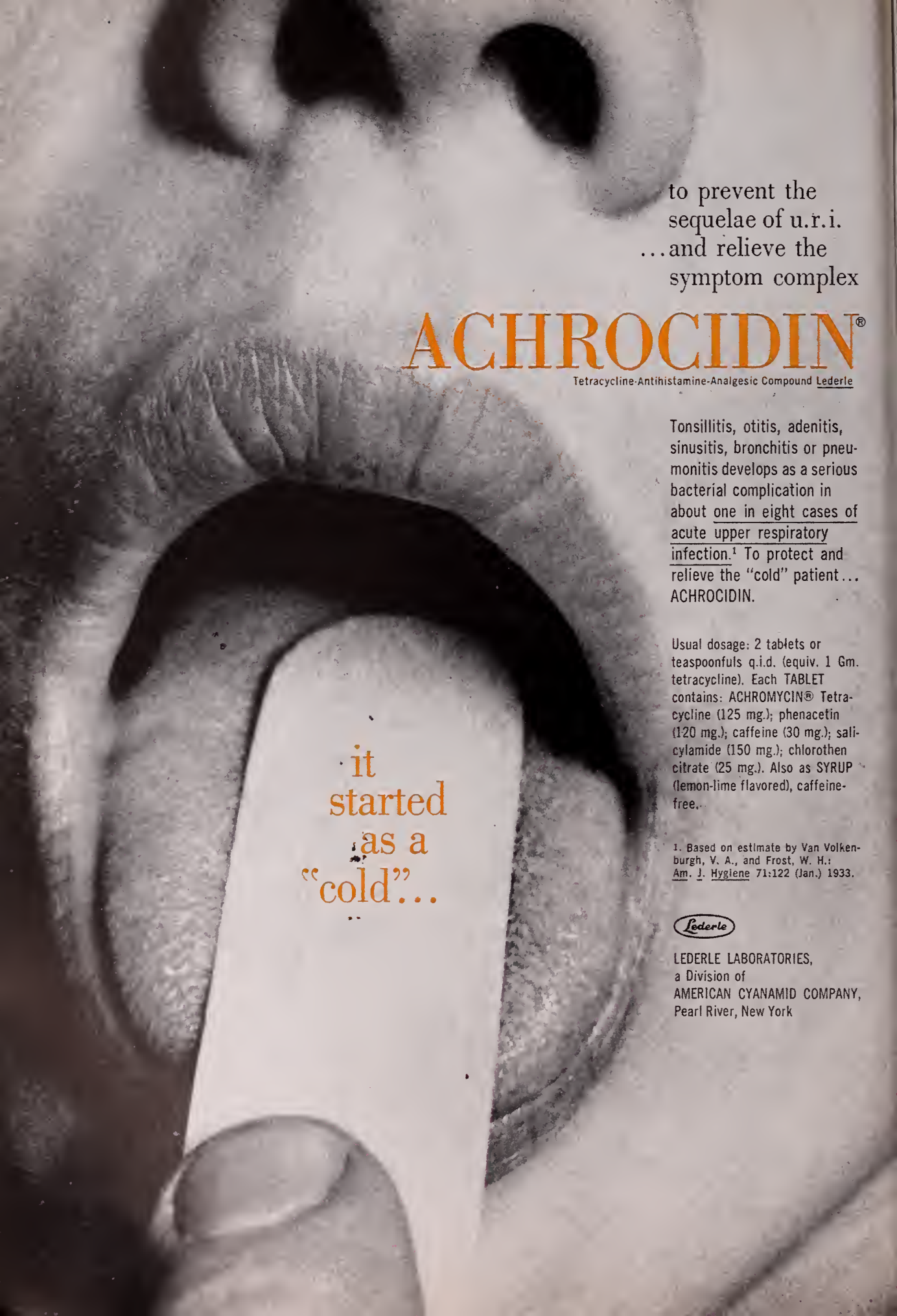
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1. Kuzell, W. C., and others.: Arch. Int. Med. 92:646, 1953. 2. Wolfson, W. Q.: J. Michigan M. Soc. 54:323, 1955. 3. Strandberg, B.: Brit. J. Phys. Med. 19:9, 1956. 4. Platt, W. D., Jr., and Steinberg, I. H.: New England J. Med. 256:823 (May 2) 1957.

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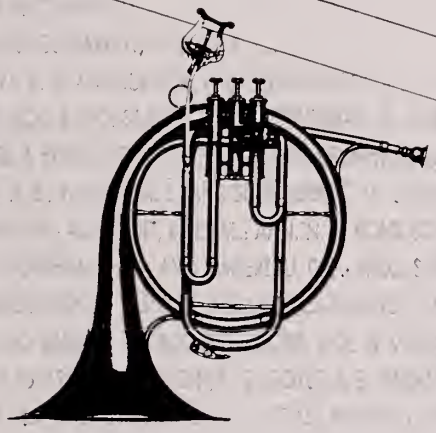
1. Based on estimate by Van Volkenburgh, V. A., and Frost, W. H.: Am. J. Hygiene 71:122 (Jan.) 1933.

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Rautensin bibliography: 1. WRIGHT, W. T., JR.; POKORY, C., AND FOSTER, T.: AM. PRACT. & DIGEST. TREAT. 7:1992, 1956. 2. SUCKLE, E.: GERIATRICS 11:509, 1956. 3. FINCH, W. J.: J. OKLAHOMA M.A. 50:259, 1957. 4. TERMAN, L. A.: ILLINOIS M. J. 3:67, 1957. 5. GIFFORD, R. W.: J. ARKANSAS M. SOC. 55:31, 1958. 6. FORD, R. V., AND MOYER, J. H.: POSTGRAD. MED. 23:41, 1958.

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Miller, R. F.: Clin. Rev. 1:10 (July) 1958

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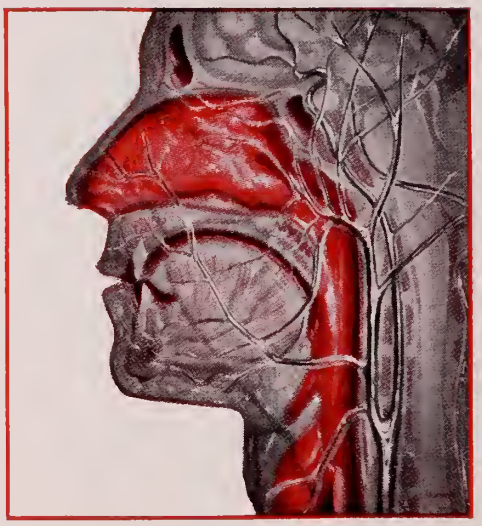
1. Lippmann, O.: Arch. Ophth. **57**:339, March 1957.
2. Gordon, D.M.: Am. J. Ophth. **46**:740, November 1958.

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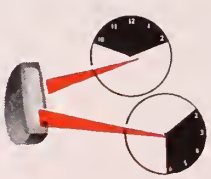
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1. Fabricant, N. D.: E.E.N.T. Monthly *37*:460 (July) 1958.
2. Lhotka, F. M.: Illinois M. J.: *112*:259 (Dec.) 1957.
3. Farmer, D. F.: Clin. Med. *5*:1183 (Sept.) 1958.

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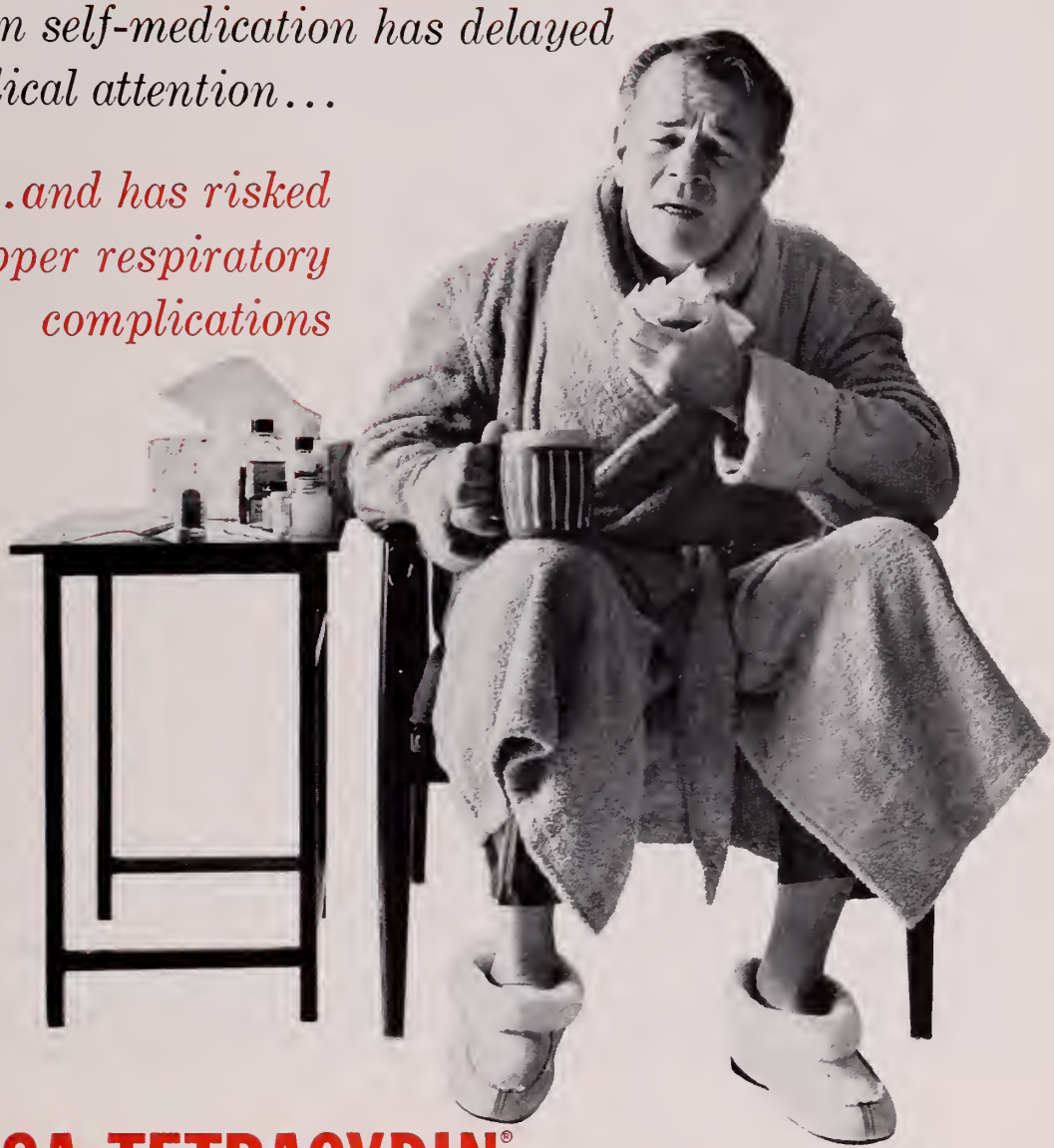
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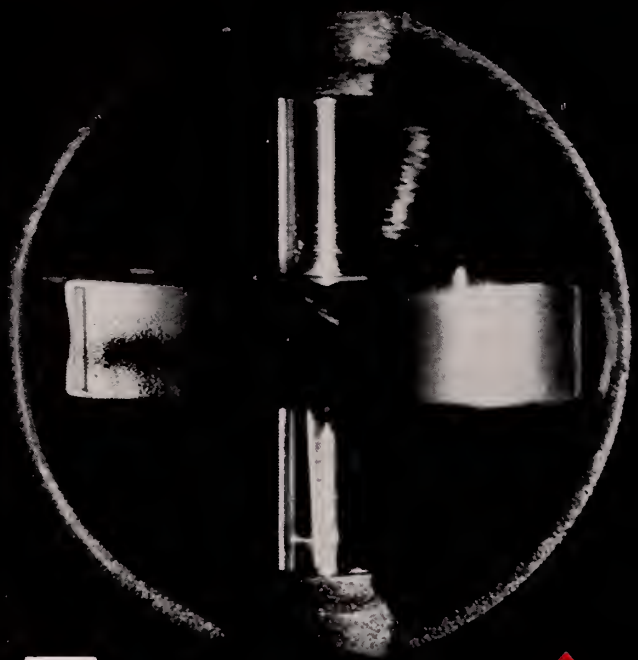
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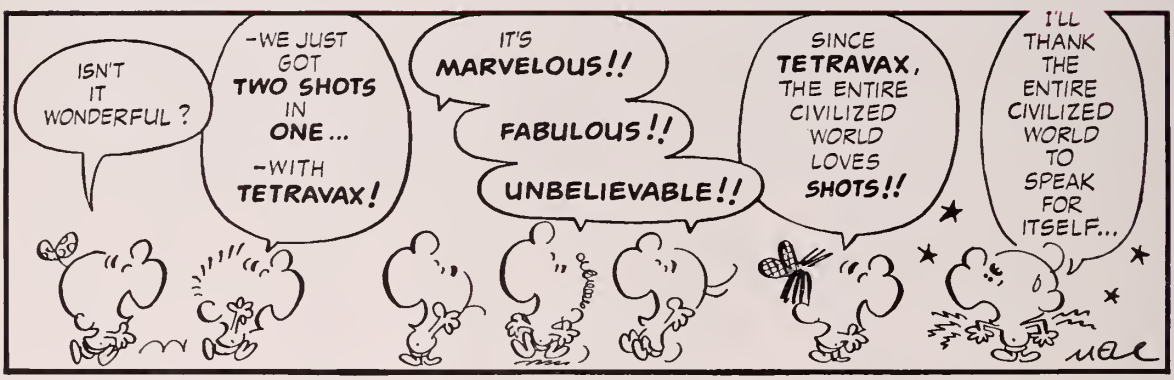
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Poliomyelitis-Diphtheria-Pertussis-Tetanus

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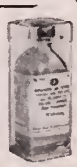


TETRAVAX®

DIPHTHERIA AND TETANUS TOXOIDS WITH PERTUSSIS AND POLIOMYELITIS VACCINES

now you can immunize against more *diseases...with* fewer *injections*

Dose: 1 cc.
Supplied: 9 cc. vials in *clear plastic* cartons. Package circular and material in vial can be examined without damaging carton. Expiration date is on vial for checking even if carton is discarded.



For additional information, write Professional Services, Merck Sharp & Dohme, West Point, Pa.

TETRAVAX IS A TRADEMARK OF MERCK & CO., INC.

MSD MERCK SHARP & DOHME, DIVISION OF MERCK & CO., INC., PHILADELPHIA 1, PA.

■ Exhibits unusual analgesic properties, different from those of any other drug ■ Specific and superior in relief of SOMAtic pain ■ Modifies central perception of pain without abolishing natural defense reflexes ■ Relaxes abnormal tension of skeletal muscle

SOMA™

N-isopropyl-2-methyl-2-propyl-1, 3-propanediol dicarbamate

■ More specific than salicylates ■ Less drastic than steroids

■ More effective than muscle relaxants

SOMA has an unique analgesic action. It apparently modifies central pain perception without abolishing peripheral pain reflexes. SOMA is particularly effective in relieving joint pain. Patients say that they feel better and sleep better with SOMA than with previously used analgesic, sedative or relaxant drugs.
SOMA also relaxes muscle hypertonia, with its stresses on related joints, ligaments and skeletal structures.

ACTS FAST. Pain-relieving and relaxant effects start in 30 minutes and last 6 hours.

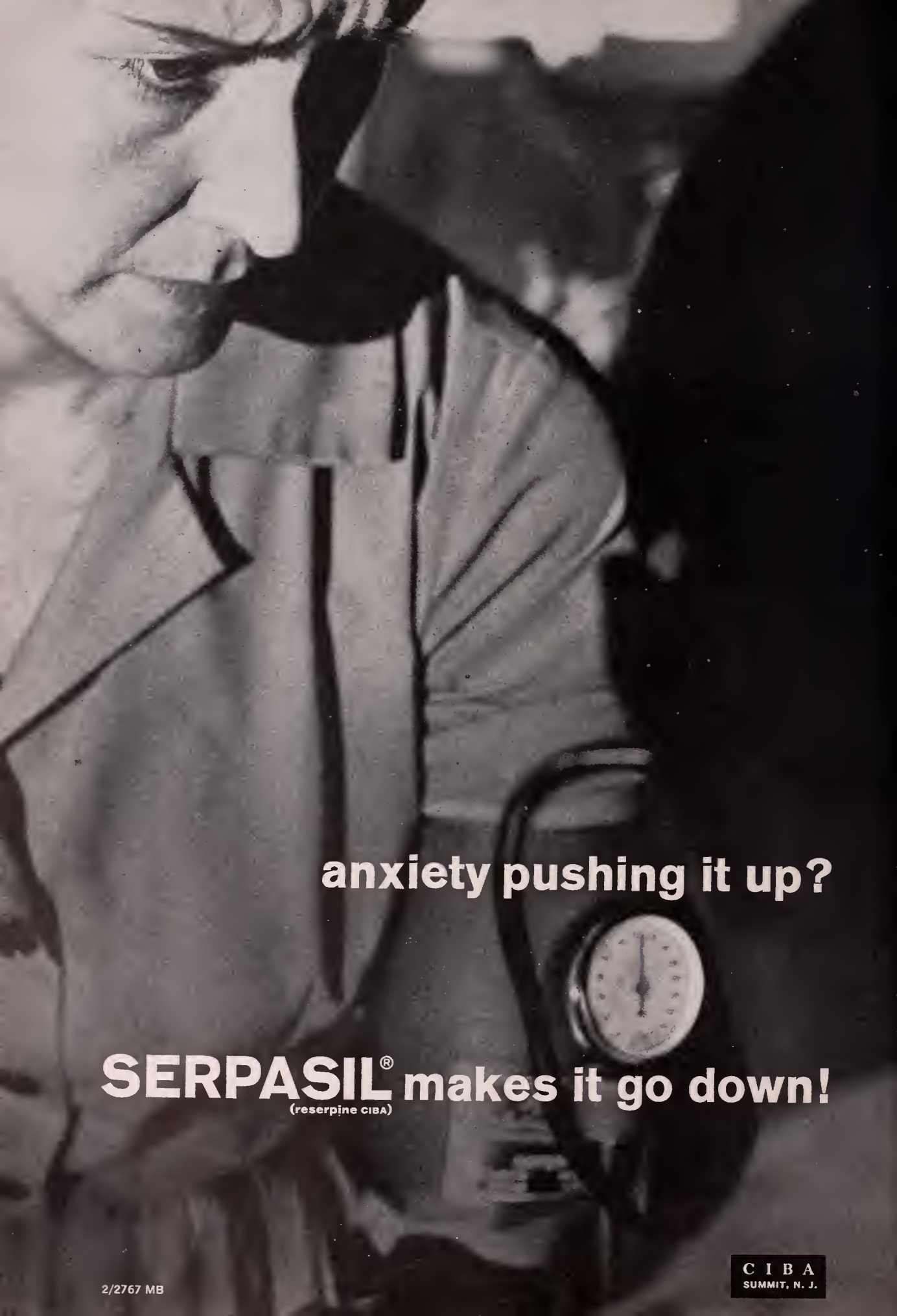
NOTABLY SAFE. Toxicity of SOMA is extremely low. No effects on liver, endocrine system, blood pressure, blood picture or urine have been reported. Some patients may become sleepy, particularly on high dosage.

EASY TO USE. Usual adult dose is one 350 mg. tablet 3 times daily and at bedtime.

SUPPLIED: Bottles of 50 white coated 350 mg. tablets.

Literature and samples on request.

WALLACE LABORATORIES, NEW BRUNSWICK, N. J.


anxiety pushing it up?
SERPASIL®
(reserpine CIBA)
makes it go down!
2/2767 MB
CIBA
SUMMIT, N. J.



Doctors, too, like "Premarin"®

THE doctor's room in the hospital is used for a variety of reasons. Most any morning, you will find the internist talking with the surgeon, the resident discussing a case with the gynecologist, or the pediatrician in for a cigarette. It's sort of a club, this room, and it's a good place to get the low-down on "Premarin" therapy.

If you listen, you'll learn not only that doctors like "Premarin," but *why* they like it.

The reasons are fairly simple. Doctors like "Premarin," in the first place, because it really relieves the symptoms of the menopause. It doesn't just mask them — it replaces what the patient lacks — natural estrogen. Furthermore, if the patient is suffering from headache, insomnia, and arthritic-like symptoms due to estrogen deficiency, "Premarin" takes care of that, too.

"Premarin," conjugated estrogens (equine), is available as tablets and liquid, and also in combination with meprobamate or methyltestosterone.

Ayerst Laboratories • New York 16, N. Y. • Montreal, Canada

Ayerst®

Where a poly-unsaturated oil is called for in the diet, **Wesson®** satisfies the most exacting requirements

(—and the most exacting appetites).



Compared to other readily available vegetable oils, Wesson is unsurpassed as a serum cholesterol depressant.

Each pint of Wesson contains 437-524 Int. Units of Vitamin E

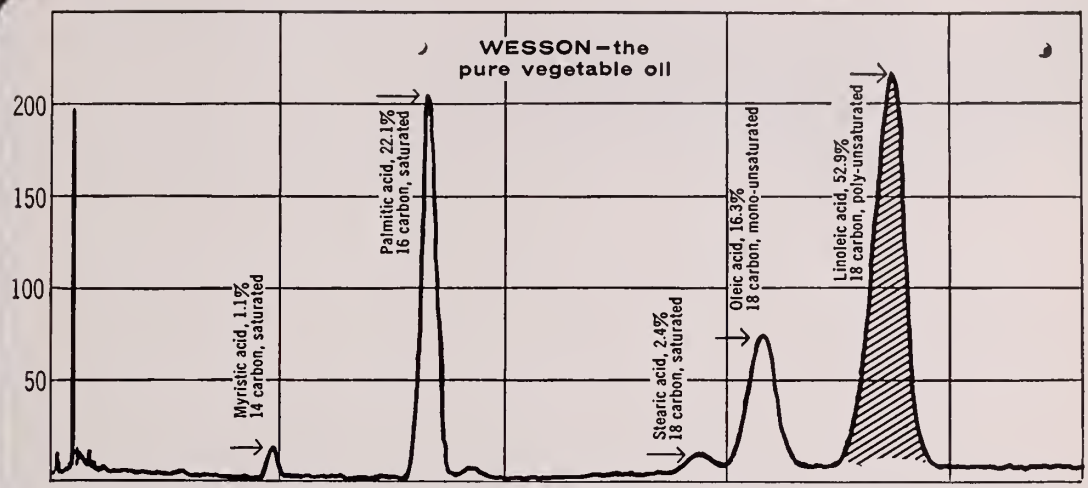
Faithful adherence to any diet is much more likely when foods taste good. The preference for Wesson—amply confirmed by its sales leadership for 59 years—has been reconfirmed in recent tests with brand identification removed. Housewives in a national probability sample indicated marked preference for Wesson, particularly by the criteria of odor, flavor (blandness) and lightness of color.

Wesson's Important Ingredients:

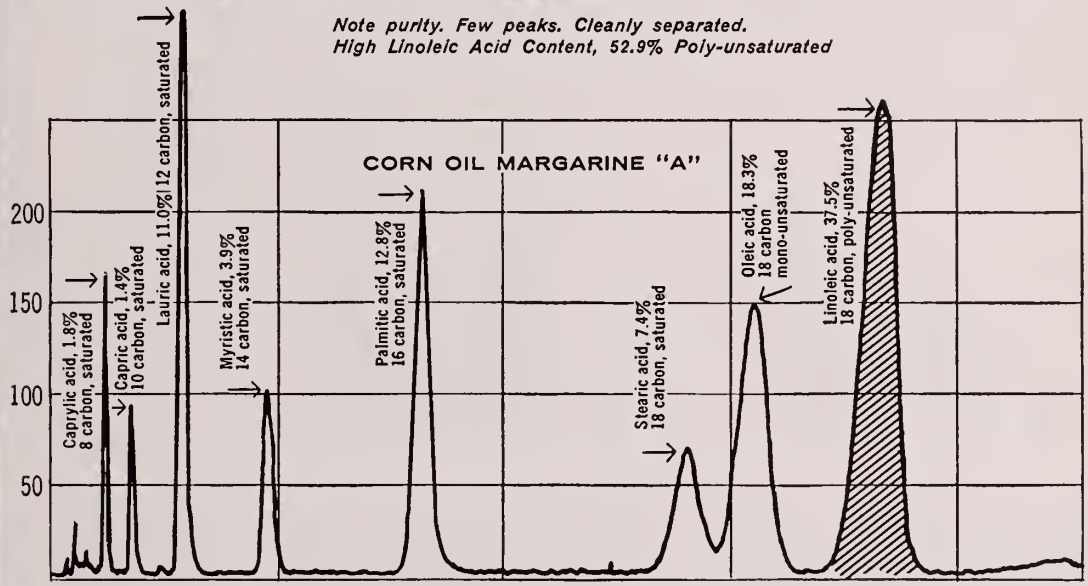
Linoleic acid glycerides	50% to 55%
Phytosterol (predominantly beta sitosterol)	0.4% to 0.7%
Total tocopherols	0.09% to 0.12%

Never hydrogenoted—completely solt free

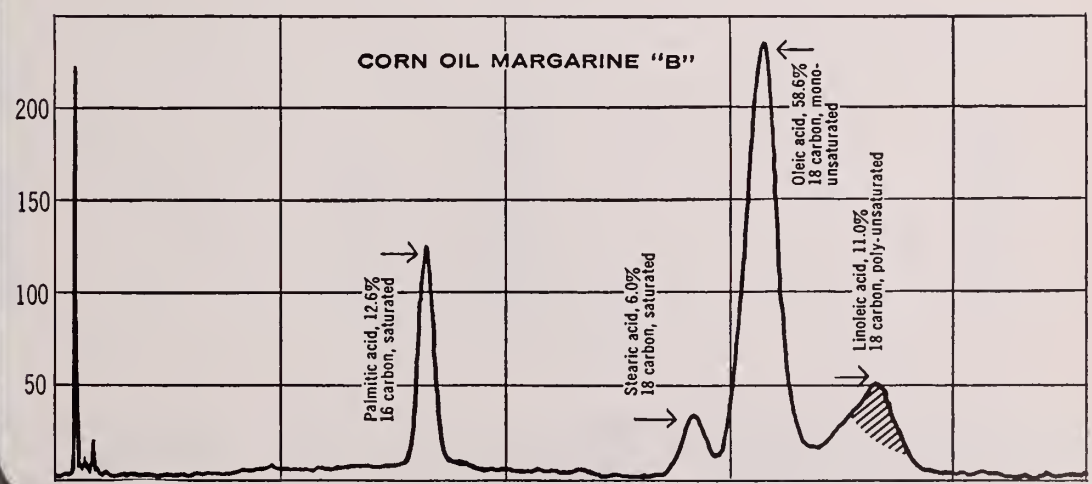
GAS CHROMATOGRAPHS *Produced by Independent Laboratory*



Note purity. Few peaks. Cleanly separated.
High Linoleic Acid Content, 52.9% Poly-unsaturated



Note effect of coconut oil. Medium Linoleic Acid Content, 37.5%



Note effect of hydrogenation. Low Linoleic Acid Content, 11%

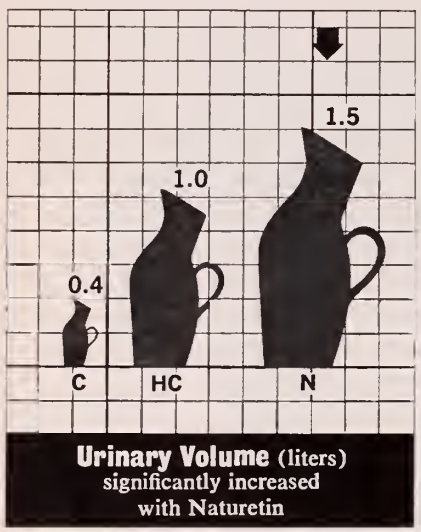
more closely approaches the ideal diuretic

Naturētin

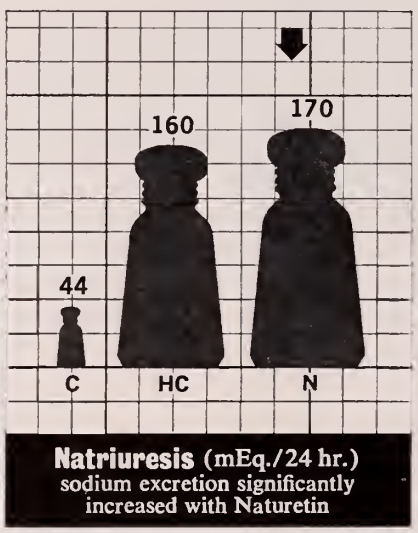
Squibb Benzydroflumethiazide

"When compared to other members of this heterocyclic group of compounds, this drug [NATURETIN] shows a significantly increased natriuresis and decreased loss of potassium and bicarbonate. In this respect it more closely approaches a natural or 'ideal diuretic.' It is effective upon continuous administration and causes no significant serum biochemical changes. It is effective in a wide variety of edematous and hypertensive states and represents a significant advance in diuretic therapy." *Ford, R.V.: Pharmacological observations on a more potent benzothiadiazine diuretic; accepted for publication by the American Heart Journal.*

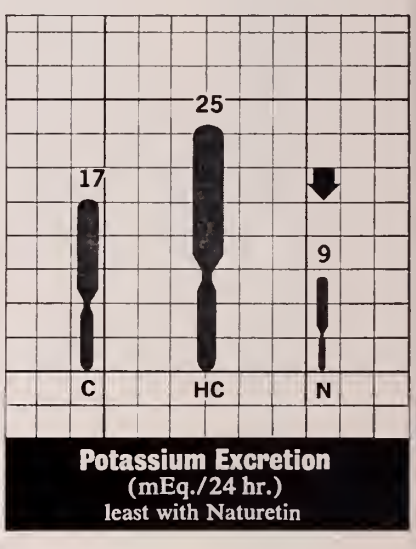
Comparison of electrolyte excretion pattern for the 24 hours following typical doses of chlorothiazide, hydrochlorothiazide, and Naturetin[1]



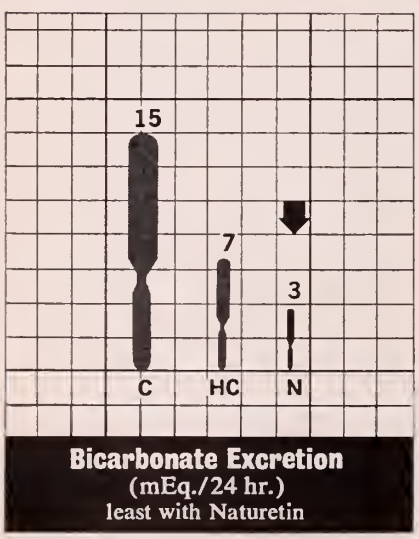
Urinary Volume (liters) significantly increased with Naturetin



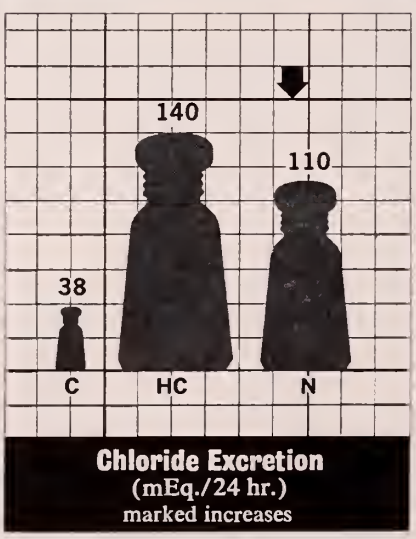
Natriuresis (mEq./24 hr.) sodium excretion significantly increased with Naturetin



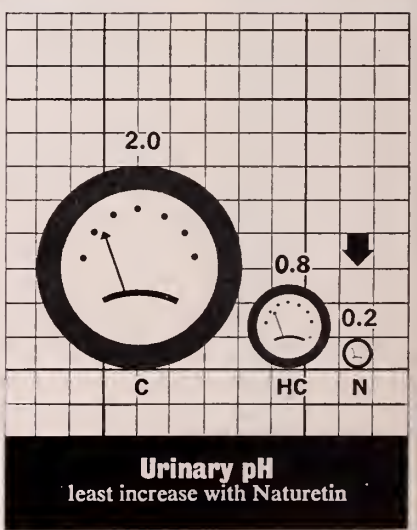
Potassium Excretion (mEq./24 hr.) least with Naturetin



Bicarbonate Excretion (mEq./24 hr.) least with Naturetin



Chloride Excretion (mEq./24 hr.) marked increases



Urinary pH least increase with Naturetin

Typical Doses: Chlorothiazide—1,000 mg.; Hydrochlorothiazide—50 mg.; Naturetin (Benzydroflumethiazide)—5 mg.

1. Adapted from: Ford, R. V., Squibb Clin. Res. Notes 2:1 (Dec.) 1959.

A single 5 mg. tablet once a day provides all these advantages[2]

- prolonged action — in excess of 18 hours
- convenient once-a-day dosage
- low daily dosage — more economical for the patient
- no significant alteration in normal electrolyte excretion pattern
- repetitively effective as a diuretic and antihypertensive
- greater potency mg. for mg.—more than 100 times as potent as chlorothiazide
- potency maintained with continued administration
- low toxicity — few side effects — low salt diets not necessary
- comparative studies with chlorothiazide, hydrochlorothiazide, and Naturetin disclose that smallest doses of Naturetin produce greater weight loss per day
- in hypertension, Naturetin, alone or in combination with other antihypertensives, produces significant decreases in mean blood pressure and other favorable clinical effects
- purpura and agranulocytosis not observed
- allergic reactions rarely observed

[2]Reports (1959) to the Squibb Institute for Medical Research.

Naturētin —*Indications:* in control of edema when diuresis is required, in congestive heart failure, in the premenstrual syndrome, nephrosis and nephritis, cirrhosis with ascites, edema induced by drugs (certain steroids); in the management of hypertension, used alone, combined with Raudixin (Squibb Rauwolfia Serpentina Whole Root), or with other antihypertensive drugs, such as ganglionic blocking agents.

Contraindications: none, except in complete renal shutdown.

Precautions: when Naturetin is added to an antihypertensive regimen including hydralazine, veratrum, and/or ganglionic blocking agents, immediate reduction must be made in the dosage for all preparations; the dosage for ganglionic blocking agents must be decreased by 50% to avoid a precipitous drop in blood pressure. This also applies if these hypotensive drugs are added to an established Naturetin regimen . . . in hypochloremic alkalosis with or without hypokalemia . . . in cirrhotic patients or those on digitalis therapy when reductions in serum potassium are noted . . . in diabetic patients or those predisposed to diabetes . . . when increased uric acid concentrations are noted . . . when signs—leg or abdominal cramps, pruritus, paresthesia, rash—suggestive of hypersensitivity, are noted.

Naturētin —*Dosage: in edema,* average dose, 5 mg., once daily, preferably in the morning; to initiate therapy, up to 20 mg., once daily or in divided doses; for maintenance, 2.5 to 5.0 mg., daily in a single dose. *In hypertension:* suggested initial dose, 5 to 20 mg. daily; for maintenance, 2.5 to 15 mg. daily, depending on the individual response of the patient. When Naturetin is added to an antihypertensive regimen with other agents, lower maintenance doses of each drug should be used.

Naturētin —*Supplied:* tablets of 2.5 mg. and 5 mg. (scored).

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Squibb Quality—the Priceless Ingredient

Rx Naturetin 5mg. #30 Disp. Sig: 1 each morning

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saturation doses – the hard way!

Each of these food portions contains a saturation dose of one of the water-soluble B vitamins or C. The easy way to provide such quantities of these vitamins with speed, safety and economy is to prescribe Allbee with C. Recommended in pregnancy, deficiency states, digestive dysfunction and convalescence.

In each Allbee with C:		As much as:*
Thiamine mononitrate (B_1)	15 mg.	6.9 lbs. of fried bacon
Riboflavin (B_2)	10 mg.	31½ ozs. of liverwurst
Pyridoxine HCl (B_6)	5 mg.	2 lbs. of yellow corn
Nicotinamide	50 mg.	11 ozs. of roasted peanuts
Calcium pantothenate	10 mg.	¼ lb. of fried beef liver
Ascorbic acid (Vitamin C)	**250 mg.**	¾ lb. of cooked broccoli

*These common foods are among the richest sources of B vitamins and ascorbic acid. H.A. Wooster, Jr., Nutritional Data, 2nd Ed., Pittsburgh, 1954.

Allbee® with C

Robins

A. H. ROBINS COMPANY, INC.
RICHMOND 20, VIRGINIA

the beauty of these antitussives:



they help the cough remove its cause

These elegant antitussives comprise a group of significantly superior expectorants from which you may select the formula best suited for your coughing patient.

First of all, they have more in common than mere delectability to eye and palate: they all include *glyceryl guaiacolate*. This remarkable expectorant aids the coughing mechanism by increasing the secretion of Respiratory Tract Fluid,[1] which helps liquefy sputum,[1,3] makes bronchial and tracheal cilia more efficient,[1,2] and acts as a demulcent.[1,3-5] Through its effects, all four expectorants promote the natural purpose of the cough, which is to remove the irritants that cause it.[1,2]

In addition, the Robins antitussive armamentarium provides a choice of widely accepted drugs in various combinations with glyceryl guaiacolate for treating different kinds of coughs and associated symptoms. For antihistaminic effects, there is Dimetane® or prophenpyridamine; for bronchodilation and nasal decongestion, there are sympathomimetic agents; and for suppression of the "too frequent" cough, there is codeine or dihydrocodeinone.

Robitussin®

Each teaspoonful contains:

Glyceryl guaiacolate ..... 100 mg.

Robitussin® A-C

Each teaspoonful contains:

Glyceryl guaiacolate ..... 100 mg.
Prophenpyridamine maleate ... 7.5 mg.
Codeine phosphate ..... 10 mg.
(exempt narcotic)

Dimetane® Expectorant

Each teaspoonful contains:

Parabromdylamine maleate (DIMETANE) ..... 2 mg.
Glyceryl guaiacolate ..... 100 mg.
Phenylephrine HCl, USP ..... 5 mg.
Phenylpropanolamine HCl, NNR ..... 5 mg.

Dimetane® Expectorant-DC

Each teaspoonful contains the Dimetane Expectorant formula *plus* Dihydrocodeinone bitartrate, NF ..... 1.8 mg.
(exempt narcotic)

References: 1. Cass, L. J., and Frederik, W. S.: Am. Pract. & Digest Treat. *2*:844, 1951. 2. Blanchard, K., and Ford, R. A.: Journal-Lancet *74*:443, 1954. 3. Hayes, E. W., and Jacobs, L. S.: Dis. Chest *30*:441, 1956. 4. Blanchard, K., and Ford, R. A.: Rocky Mountain M. J., Vol. 52, No. 3, 1955. 5. Boyd, E. M., and Pearson: Am. J. M. Sc. *211*:602, 1946.

Robins

A. H. ROBINS COMPANY, INC., RICHMOND 20, VIRGINIA

This is Panalba performance...





in sinusitis

. . . into a mixed culture of the four organisms commonly involved in sinusitis . . . *Str. hemolyticus, D. pneumoniae, H. influenzae* and *Staph. aureus* (in this case a resistant strain) . . . we introduce the five most frequently used antibiotics.

Twenty-four hours later (in this greatly enlarged photograph), note that only *one* of the five leading antibiotics has stopped *all* the organisms, including the resistant staph! This is Panalba.

In your next patient with sinusitis . . . in *all* your patients with potentially-serious infections . . . provide this extra protection with your prescription:

Dosage—1 or 2 capsules 3 or 4 times a day.
Supplied—Capsules containing Panmycin phosphate equivalent to 250 mg. tetracycline hydrochloride, and 125 mg. Albamycin as novobiocin sodium, in bottles of 16 and 100.
Now available: **new Panalba Half-Strength Capsules in bottles of 16 and 100.**

Panalba*

(Panmycin* Phosphate plus Albamycin*)

The broad-spectrum antibiotic of *first* resort



Upjohn The Upjohn Company
Kalamazoo, Michigan

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RELA™

CARISOPRODOL

EASES STRAINS SPRAINS & LOW BACK PAINS...!

X



RELA—a new myogesic for better relaxant *and* analgesic therapy—more adept management of spasm and pain in strains, sprains and low back pains.

RELA—though a single drug—is a true myogesic and works rapidly to achieve three desired effects...

Rela relaxes acute muscle spasm
Relief of muscle spasm (96% excellent to good effectiveness)[1]

Rela provides a unique quality of persistent pain relief through its relaxant and analgesic actions
"Relief from pain was usually rapid and sometimes dramatic"[1]

Rela, through relaxation and analgesia, assures daytime ease and nighttime rest
"... A number of patients reported freedom from insomnia which they attributed to freedom from pain."[1]

indications: RELA is most beneficial in those conditions of the musculoskeletal system manifesting pain, stiffness and spasm.

safety: Studies of more than 1400 patients indicate that the toxicity of RELA is exceptionally low. In human subjects, respiratory, blood pressure or blood chemistry changes and/or renal, hepatic or endocrine dysfunction have not been reported.

dosage: The usual adult dosage of RELA is one tablet 3 times daily and at bedtime. RELA has a rapid onset of action, with relief usually apparent within 30 minutes, and persisting for at least 6 hours.

supply: RELA is available as 350 mg., pink, coated tablets in bottles of 30.

1. Kuge, T.: To be published.

H-227

MYOGESIC
muscle-analgesic relaxant

Schering

A SPOON LICKIN' GOOD SULFA!



it's delicious cherry-flavored for children

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N[1] Acetyl Sulfamethoxypyridazine Lederle

just 1 dose a day . . . achieves rapid therapeutic levels . . . sustained for 24 hours . . . extremely low incidence of sensitivity reactions and renal complications . . . convenient, highly economical . . .

ALWAYS ACCEPTABLE . . . WHENEVER SULFAS ARE INDICATED

Recommended dosage: first-day dose is 1 teaspoonful (250 mg.) for each 20 lbs. body weight up to 80 lbs. For each day thereafter, ½ teaspoonful for each 20 lbs. For 80 lbs. and over, use adult dosage of 4 teaspoonfuls (1.0 Gm.) initially, and 2 teaspoonfuls (0.5 Gm.) daily thereafter. Administer immediately after a meal.

Supplied: Each teaspoonful (5 cc.) contains 250 mg. of sulfamethoxypyridazine activity. Bottles of 4 and 16 fl. oz.

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—whether it is in connection with policy changes, with filing of a claim or supplying you with information concerning your coverages.

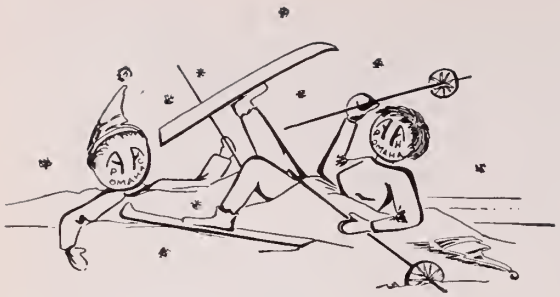
Service is essential to effective operation and success of your OSMA insurance program.

Other important features of your OSMA plan: Flexible, modern, up-to-date coverages; flexible monthly benefits ranging from $200 to $600, according to underwriting agreement; choice of three-year or five-year illness coverage; choice of three waiting periods.

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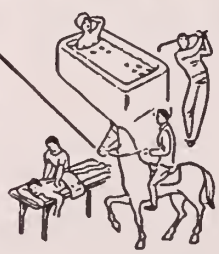
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seem to crowd
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into manageable order.

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NEW
MYOGESIC[X]

RELA™ – EASES MUSCLE SPASM & PAIN IN SPRAINS, STRAINS, LOW BACK PAINS

X

CARISOPRODOL

[X]MYOGESIC
muscle relaxant – analgesic

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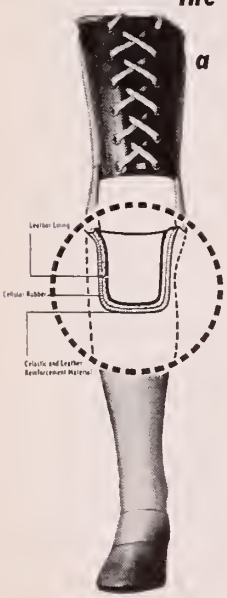
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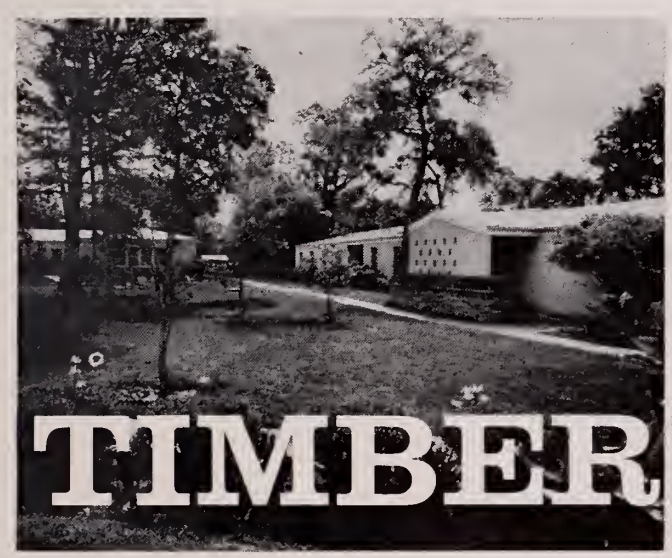
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DAY HOSPITAL

DEPARTMENT OF OUT PATIENT PSYCHIATRY

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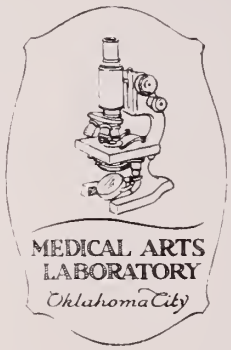
Narcotic Cases Not Admitted

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Index to Advertisers

Abbott Laboratories ..... xvi, xxiii
Ames Company, Inc. ..... inside back
Arlington Hotel xlvi
Armour Pharmaceutical Co. xv
Ayerst Laboratories xxxiii

Beverly Hills Clinic lvii
Bone and Joint Hospital li
Bristol Laboratories, Inc. ..... insert
Burroughs-Wellcome & Company, Inc. ..... insert, xxiv

Chicago Medical Society xxxviii
Ciba ..... vii, xxxii
Coca Cola Bottlers xlvii
Corn Products Refining Co. ..... xxxiv and xxxv
Coyne Campbell Sanitarium li
Credit Service xlvi

Geigy xvii
Glenbrook Labs xxii
Goldfain Laboratory lii

J. E. Hanger xlviii

Drs. Lain, Lamb and Jones lii
Lederle Laboratories ..... vi, xi, 48 and 49,, insert, xviii, xxviii and xxix, xliv
Eli Lilly & Company xiv

Medical Arts Laboratory l
Melton Co., Inc. xlviii
Merck Sharp and Dohme, Inc. ..... iv, xxv, xxx
McAlester Clinic liii
Midwest Surgical Supply Co., Inc. lvii

The Neurological Hospital lvi
North American Accident Insurance Co. xlv

Oklahoma Allergy Clinic liii
Osler Radioisotope Laboratory lvi

Parke Davis and Company ..... inside front and iii
Pfizer Paboratories ..... viii, xxi, xxvii
Physicians Casualty & Health Assn. xlvi

A. H. Robins Co., Inc. ..... ix, insert, xxxix
Rock Haven Hospital lvi
Roerig ..... xii, xx

St. Paul Companies xlix
G. D. Searle & Company 37
Schering Corporation ..... xiii, xxxviii, xlii and xliii, xlvii
Seven-Up Bottlers xlviii
Smith-Dorsey ..... x, xix, xxvi
Smith, Kline & French Laboratories ..... back cover
Springer Clinic liv
E. R. Squibb and Sons ..... xxxvi and xxxvii
Stout Clinical Laboratory liv
Sugg Clinic lv

Terrell's Laboratories lvi
Timberlawn Sanitarium xlix
Tulsa Clinic lv

The Upjohn Company ..... xl and xli

Wallace Laboratories ..... insert, xxxi
Winthrop Laboratories ..... inserts

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of the

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Lethargy	14	12.7
Enuresis	7	6.4
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Hypoglycemia	2	1.8
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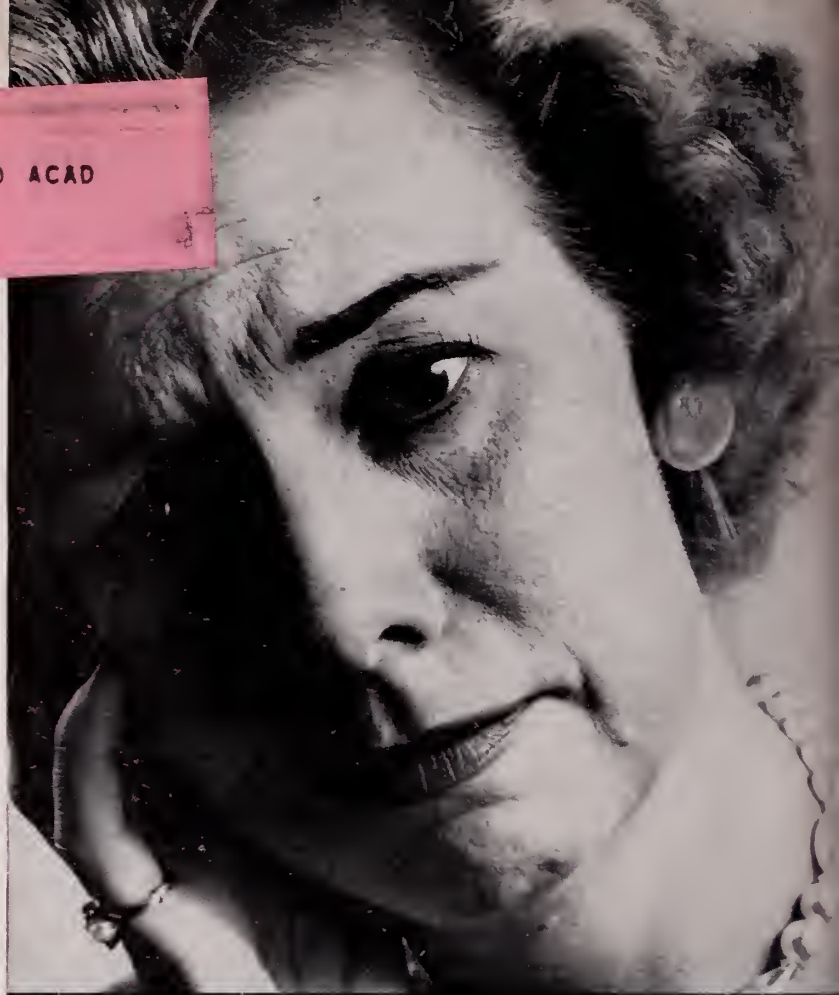
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Page 102

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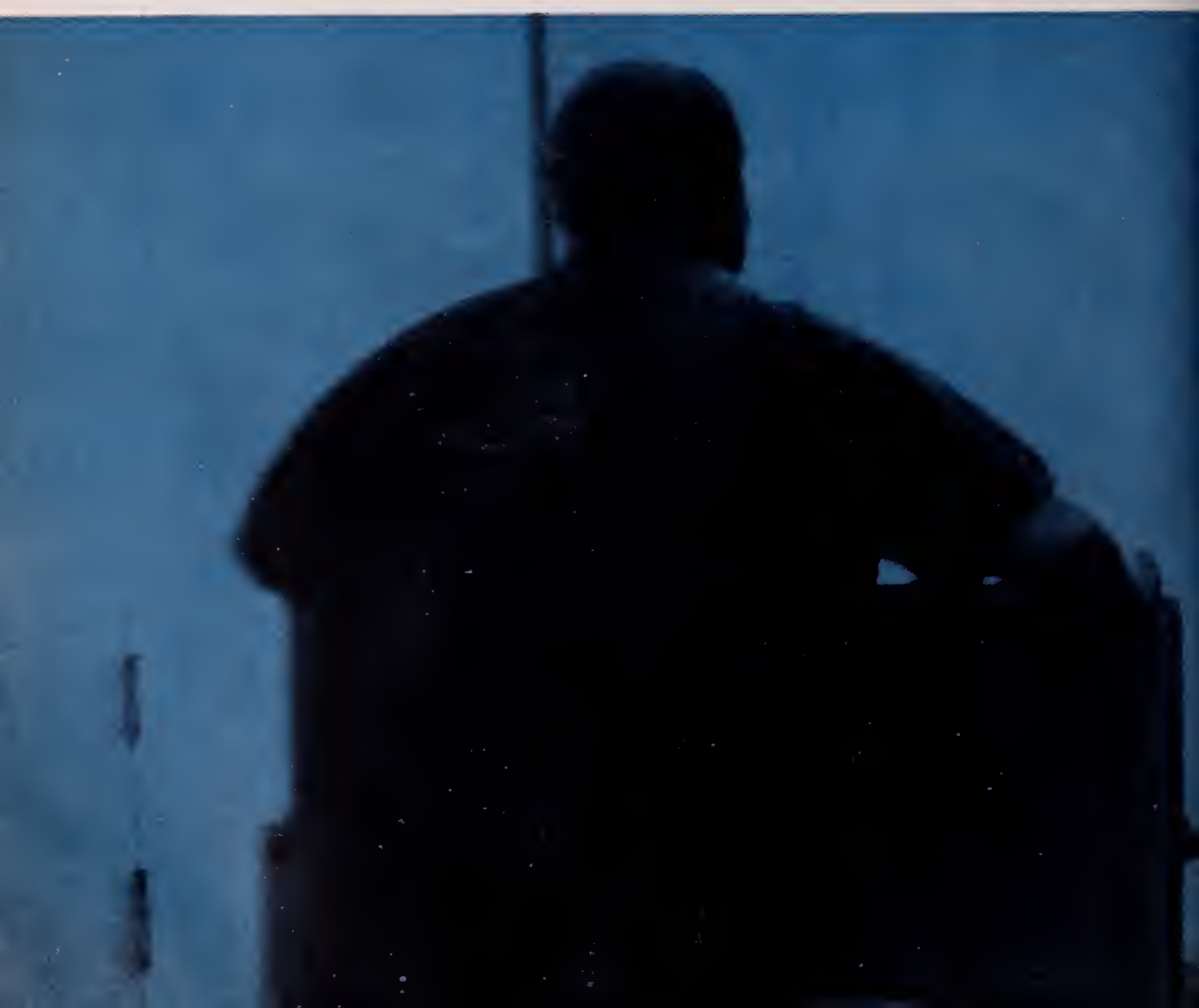
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CONTENTS

EDITORIALS

Where There Is No Vision— 61
For Doctors Only? 62
President's Letter 102

SCIENTIFIC ARTICLES

Growth Failure in Infancy and Childhood, *A Symposium* 63
Thoracic Lung Hernia, *Ira Ross Grimes, M.D., and Warren L. Felton, II, M.D.* 78
Hypothermic Total Body Cardiopulmonary Bypass, Report of 19 Consecutive Cases, *Nazih Zuhdi, M.D., Allen Greer, M.D., John Montroy, M.D., John Carey, M.D. and Bill McCollough, M.D.* 83

MEDICAL CENTER

The Present Status of the Internship 86
The Normal Radiology of Old Age and Its Borderlines, *Ernest Lachman, M.D.* 87
Etiology of Peptic Ulcer Disease, *Medical Grand Rounds* 93
Abstracts 100
Appointments Announced by Board of Regents . . . 101

SPECIAL ARTICLE

Factors Relating to Physician Distribution in Oklahoma, 1950-1959, *Robert C. Lowe, M.D., James A. Hagans, M.D. and Pearl D. Fisher, Ph.D.* 104

NEWS

Top Talent Featured for Annual Meeting 106
Oklahomans Participate in Texas Law-Science Week . 107
"Medicare" Expands Scope of Care 108
Keogh Legislation Faces Crisis 109
Third Oklahoma Colloquy Planned 110
AAGP Will Hold 12th Annual Scientific Assembly . . 112
Medical Center Presents Service Awards 112
OU Medical Research Building Underway 114
Linkletter Honors GP of the Year 120
Course on Cardiac Patient Held 121
Twenty-five Years Ago 121
Letters 122
Have You Heard? 123
Coming Meetings 124
Auxiliary News 125
Physicians Placement 126
Deaths 127
Miscellaneous Advertisements 128
Advertising Index, lxx

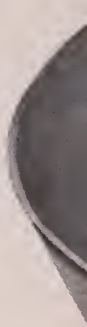
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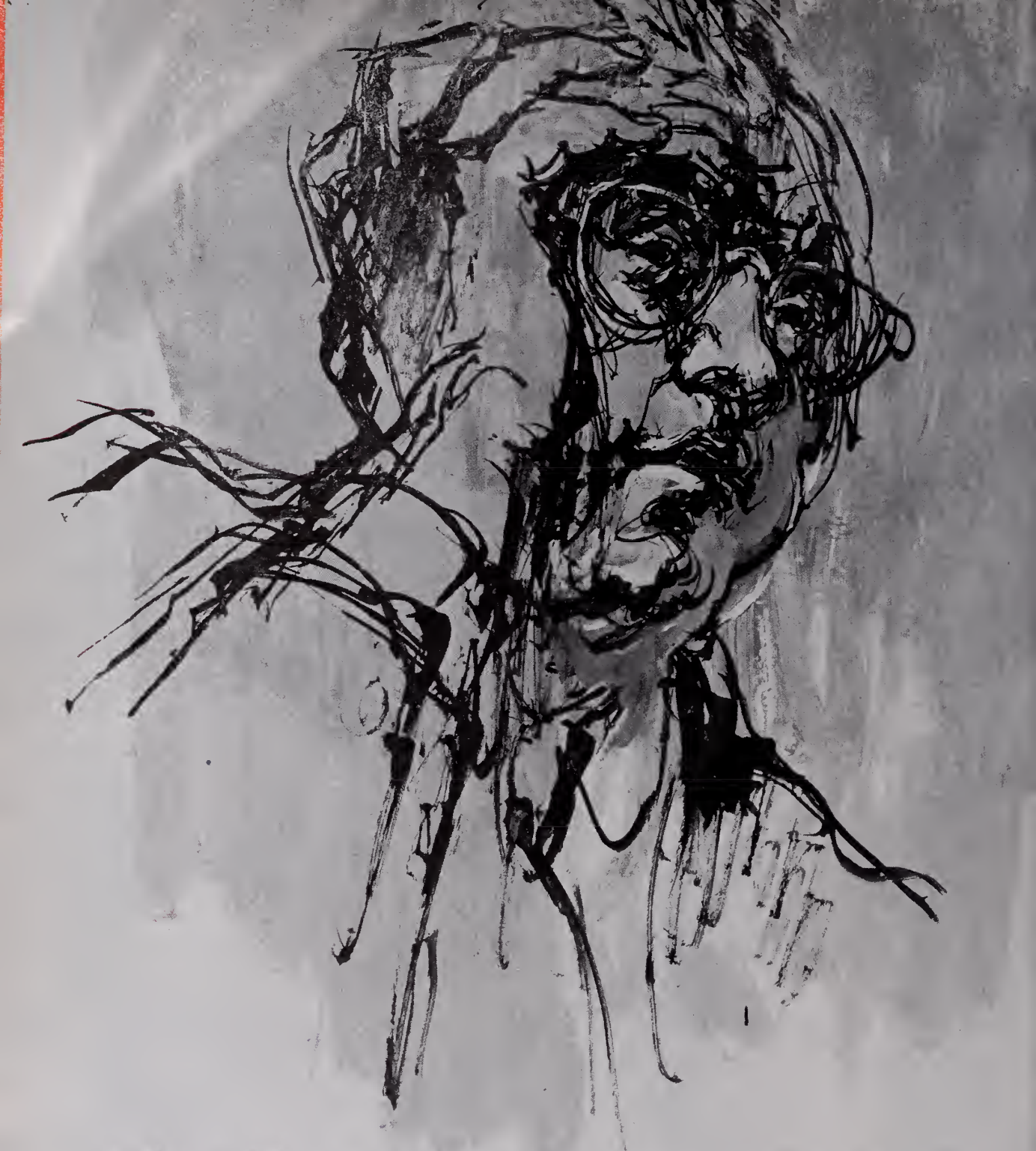
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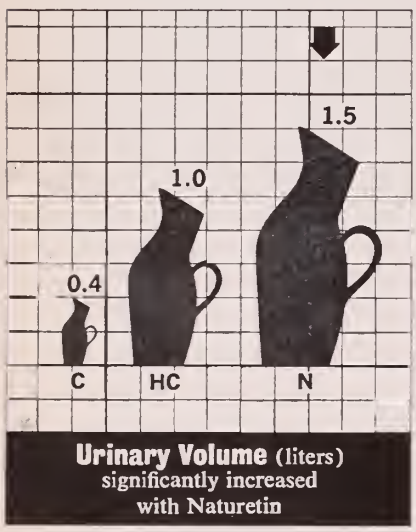
more closely approaches the ideal diuretic

Naturētin

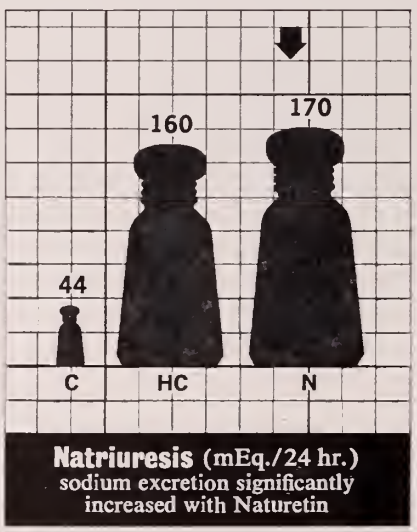
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"When compared to other members of this heterocyclic group of compounds, this drug [NATURETIN] shows a significantly increased natriuresis and decreased loss of potassium and bicarbonate. In this respect it more closely approaches a natural or 'ideal diuretic.' It is effective upon continuous administration and causes no significant serum biochemical changes. It is effective in a wide variety of edematous and hypertensive states and represents a significant advance in diuretic therapy." *Ford, R.V.: Pharmacological observations on a more potent benzothiadiazine diuretic; accepted for publication by the American Heart Journal.*

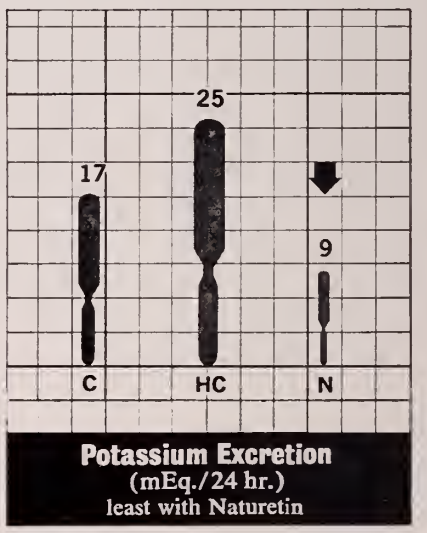
Comparison of electrolyte excretion pattern for the 24 hours following typical doses of chlorothiazide, hydrochlorothiazide, and Naturetin[1]



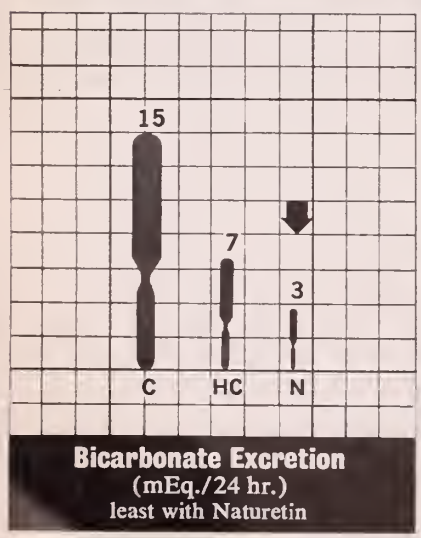
Urinary Volume (liters)
significantly increased
with Naturetin



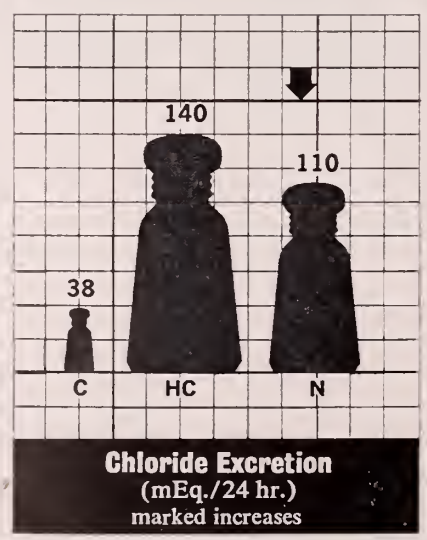
Natriuresis (mEq./24 hr.)
sodium excretion significantly
increased with Naturetin



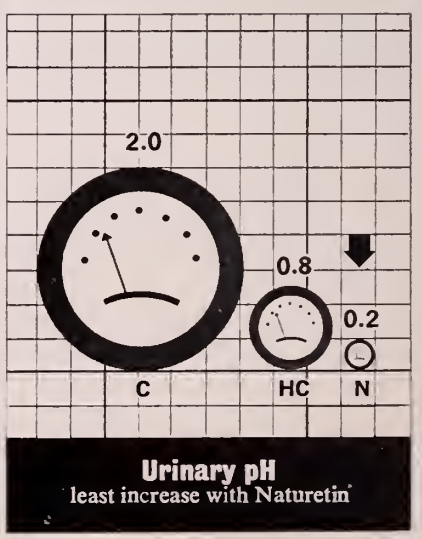
Potassium Excretion
(mEq./24 hr.)
least with Naturetin



Bicarbonate Excretion
(mEq./24 hr.)
least with Naturetin



Chloride Excretion
(mEq./24 hr.)
marked increases



Urinary pH
least increase with Naturetin

Typical Doses: Chlorothiazide—1,000 mg.; Hydrochlorothiazide—50 mg.; Naturetin (Benzydroflumethiazide)—5 mg.

1. Adapted from: Ford, R. V., Squibb Clin. Res. Notes 2:1 (Dec.) 1959.

A single 5 mg. tablet once a day provides all these advantages[2]

- prolonged action — in excess of 18 hours
- convenient once-a-day dosage
- low daily dosage — more economical for the patient
- no significant alteration in normal electrolyte excretion pattern
- repetitively effective as a diuretic and antihypertensive
- greater potency mg. for mg.—more than 100 times as potent as chlorothiazide
- potency maintained with continued administration
- low toxicity — few side effects — low salt diets not necessary
- comparative studies with chlorothiazide, hydrochlorothiazide, and Naturetin disclose that smallest doses of Naturetin produce greater weight loss per day
- in hypertension, Naturetin, alone or in combination with other antihypertensives, produces significant decreases in mean blood pressure and other favorable clinical effects
- purpura and agranulocytosis not observed
- allergic reactions rarely observed

[2]Reports (1959) to the Squibb Institute for Medical Research.

Naturētin —*Indications:* in control of edema when diuresis is required, in congestive heart failure, in the premenstrual syndrome, nephrosis and nephritis, cirrhosis with ascites, edema induced by drugs (certain steroids); in the management of hypertension, used alone, combined with Raudixin (Squibb Rauwolfia Serpentina Whole Root), or with other antihypertensive drugs, such as ganglionic blocking agents.

Contraindications: none, except in complete renal shutdown.

Precautions: when Naturetin is added to an antihypertensive regimen including hydralazine, veratrum, and/or ganglionic blocking agents, immediate reduction must be made in the dosage for all preparations; the dosage for ganglionic blocking agents must be decreased by 50% to avoid a precipitous drop in blood pressure. This also applies if these hypotensive drugs are added to an established Naturetin regimen . . . in hypochloremic alkalosis with or without hypokalemia . . . in cirrhotic patients or those on digitalis therapy when reductions in serum potassium are noted . . . in diabetic patients or those predisposed to diabetes . . . when increased uric acid concentrations are noted . . . when signs—leg or abdominal cramps, pruritus, paresthesia, rash—suggestive of hypersensitivity, are noted.

Naturētin —*Dosage: in edema,* average dose, 5 mg., once daily, preferably in the morning; to initiate therapy, up to 20 mg., once daily or in divided doses; for maintenance, 2.5 to 5.0 mg., daily in a single dose. *In hypertension:* suggested initial dose, 5 to 20 mg. daily; for maintenance, 2.5 to 15 mg. daily, depending on the individual response of the patient. When Naturetin is added to an antihypertensive regimen with other agents, lower maintenance doses of each drug should be used.

Naturētin —*Supplied:* tablets of 2.5 mg. and 5 mg. (scored).

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1. Smith, I. M., and Soderstrom, W. H.: J. A. M. A., *170*:184 (May 9), 1959.

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Editorial

Where There Is No Vision—

Are good football players more important to our state than intelligent well trained physicians? It would seem so, or at least circumstance would warrant this assumption. In our state youngsters talented toward athletics have a far easier time financing their education that those seemingly gifted toward the profession of medicine; many liberal scholarships are offered in athletics, but a student who aspires to a career in medicine finds the financial door to his ambition almost closed. At the present the medical school has but two $500.00 tuitional scholarships which they can offer to outstanding freshmen student applicants. How much money there is available for the training of students in football is anyone's guess!

The majority of freshmen students in the medical school must depend upon parental aid in order to finance their way through their training. Lacking this, their only alternative is to borrow money. As a consequence most medical students are burdened with comparatively large interest carrying debts upon graduation and which they must pay back when they enter into practice. Most medical students are compelled to borrow because the majority of them comes from families of rather slender financial means; a recent survey revealed that the parents of most medical students have an income of less than $8,000.00 a year, a sum which does not permit much latitude in financing a medical education for an ambitious son or daughter.

Our neighbor states, Kansas and Texas, appear to manifest a better sense of values than we exhibit so far as encouraging superior students into the career of medicine. Their colleges not only seem able to afford the luxury of good football teams but they appear enlightened enough to see that substantial scholarships are available for promising students who hope to become doctors. Because they attract good students into their schools the quality of medical care which their future citizens will enjoy is being upgraded.

Last year the Oklahoma School of Medicine to meet its class quota for freshmen medical students was forced to accept all but five of the qualified student applicants; some had only minimal qualifications for entrance. Unfortunately, the admission board was compelled to deny a number of extremely desirable applicants admission because these students could not obtain the necessary money required for a tuitional fee. As a result, although all these students were citizens of our state they were compelled to enroll into adjacent state schools wherein scholarships were available. It may be reasonably predicted that those students who left our state because of insufficient funds and had to go to other states to obtain their professional education will in all likelihood remain in those states for subsequent practice. Can the State of Oklahoma afford this loss?

Another aspect to this whole problem of scholarships may be noted. Top students of highschool classes who desire to enter into the various science vocations—chemistry, physics and even teaching—have a multiplicity of financial sources to whom they may apply for help: The National Science Foundation, National Institute of Health, National Defense Organization and many others, the list is long! As a result the number of admissions into these vocations has increased fourfold. Medicine, on the other hand is losing ground so far as the number of desirable applicants to the profession. Each year, although there is a demand for more physicians and the demand is becoming more critical each year, the list of students who choose a medical career is decreasing. The chances are that this is because the financial burden imposed upon students for them to obtain a medical education has become too great. A strange paradox; medicine for long has had the repute of being the most remunerative of the profession. It would appear in our society wherein so much stress is placed on money as being

the measure of success that our medical school would be overwhelmed with applicants rather than having to "scrape the barrel" as they do today. But admission boards have no alternative, they must accept students of a lesser degree of intelligence in order to meet the student quota set for the school.

Indeed, this is a lamentable commentary on our society—one where not scholastic superiority determines whether a student becomes a physician but primarily does he have the money to pay for his education!

One can be sure when these facts become generally understood by the public at large the reaction aroused may be predicted "let the government take over; why not subsidize the education of medical students by the government?" And if this situation continues this is what will actually occur, the government will subsidize medical education. Yet, every thinking person realizes that if the government controls medical education obviously they must control medical practice. Is this the backdoor route to complete socialization of the profession? It seems likely!

How to correct the problem? The only solution is for private enterprise to make available more scholarship funds in order to attract students of excellence into the profession. If this be accomplished our medical school will be in a position to select a better quality of student to fill the class rolls.

Recently a committee from the Faculty Association of the Medical School discussed this problem and came to the realization of how critical it was. A foundation plan was proposed and first steps were taken toward its organization. This foundation is a means whereby contributions from private sources may be accepted to provide more medical scholarships for freshmen students. If you feel inclined to help toward a solution of this problem may I suggest you write to Doctor Leonard P. Eliel of the Faculty Association at the Research Foundation for particulars. Possibly you may be even inclined to contribute something now, even a token amount would be helpful. If every doctor in the state would contribute one dollar a year it would be sufficient to make up a fund for three or four more exceptional students to be added to the next year class. The Faculty Association hopes by the beginning of the next scholastic year to have as much as $2,000 available for this purpose.

Are you convinced that good practitioners of medicine are as important to our state as football players? A small contribution, even a token amount, to this fund would give proof of your conviction.—*D.W.B.*

For Doctors Only?

Over the years, physicians have been contacted by various insurance companies in regard to insurance programs fraudulently proclaiming or implying association endorsement. Such statements as "Sold Only to Members of the Oklahoma State Medical Association" or, "For Doctors Only" mislead the physician-buyer into believing that he is enjoying special rates and privileges. The insurance itself may be good or bad, depending upon the underwriting company, but it cannot compete dollar for dollar with the bona-fide group programs established and administered by the association.

Currently, health and accident insurance is offered through the North American Accident Insurance Company and life insurance through the Massachusetts Mutual Life Insurance Company. The former is administered through the Insurance Committee and the latter through the Oklahoma State Medical Association Insurance Trust, a group comprised of the association President, President-Elect and Executive Secretary.

Both of the plans were designed to offer group-rated premiums, stability and benefits tailored to the needs of of most physicians.

Individual policies may suit a specific instance as well as the group plans, but don't be misled by misrepresentative claims of association endorsement.

Scientific Articles

A Symposium

Growth Failure in Infancy and Childhood*

INTRODUCTION: Doctor Riley

THE MAIN BUSINESS of a child is healthy growth and development. Because many disease processes affect them adversely, growth failure may furnish the first clue that disease is present. True and suspected growth failure are among the most frequent problems encountered by the practitioner who sees children.

When presented with a patient with growth failure, the physician must first determine the validity of the diagnosis. A large percentage of the children whom the average pediatrician sees because of this complaint have nothing wrong with them. Parents do not generally appreciate the normal slowing of fetal growth and other physiologic aspects of normal growth. Norms for physical measurement such as height and weight are easily obtained by lay persons and applied anxiously to their children without the realization that these are averages which do not allow for normal deviation. Only the physician can determine if the complaint of growth failure is valid.

The so-called average height or weight value for a particular age is a misleading index since it is usually only a lucky coincidence that the child's height or weight will match the average figure. Far more adequate is the percentile chart which is derived from the normal distribution bell-shaped curve. In percentile charts, the mean is the fiftieth percentile; the mean plus and minus two standard deviations respectively generally covers the biologic variation from the 3rd to the 97th of random sampling. While an individual whose measurements fall within these limits may be affected by disease, most measurements included within these two limits are generally considered statistically within normal range. Such charts clearly show the wide spread between children whose weight or height is low normal and those children in whom height or weight is high normal. Growth can be regarded as abnormal in most cases when repeated measurements are found to be outside of these limits. Most healthy children tend to follow the same growth channel as they grow older.

In order to utilize adequately such percentil charts, it is highly desirable to record serial measurements made at intervale appropriate to the child's age rather than at-

PROGRAM

Introduction—Harris D. Riley, Jr., M.D., *Professor of Pediatrics*

Endocrine Aspects—Doman K. Keele, M.D., *Assistant Professor of Pediatrics*

Metabolic Aspects—H. Neil Kirkman, M.D., *Assistant Professor of Pediatrics*

Neurological Aspects—Lucius Waites, M.D., *Assistant Professor of Pediatrics and Neurology*

Psychologic, Sensory and Perceptual Aspects—Sylvia Richardson, M.D., *Assistant Professor of Pediatrics*

Cardiac Aspects—Glen G. Cayler, M.D., *Assistant Professor of Pediatrics*

Infectious Aspects—Harris D. Riley, Jr., M.D., *Professor of Pediatrics*

*From the Department of Pediatrics and the Children's Memorial Hospital, University of Oklahoma School of Medicine, Oklahoma City. Presented at the Dedicatory Meeting, Mabee Children's Hospital, Tulsa, Oklahoma, September 19, 1959.

tempt to use a single set of physical measurements which may be misleading. Figure 1 portrays the erroneous impression which may result from using only a single weight measurement. At 9½ months of age, both Patients A and B weighed essentially the same. However, it is very obvious that Patient B, who weighed only four pounds, two ounces, at birth, has demonstrated an excellent growth response and is now within normal percentile channels. On the other hand, Patient A, who weighed eight pounds at birth, has not gained adequately since six months and is an example of growth failure although his weight at 9½ months was the same as Patient B. The cause of the growth failure was found to be pyelonephritis superimposed on an obstructive lesion of the urinary tract.

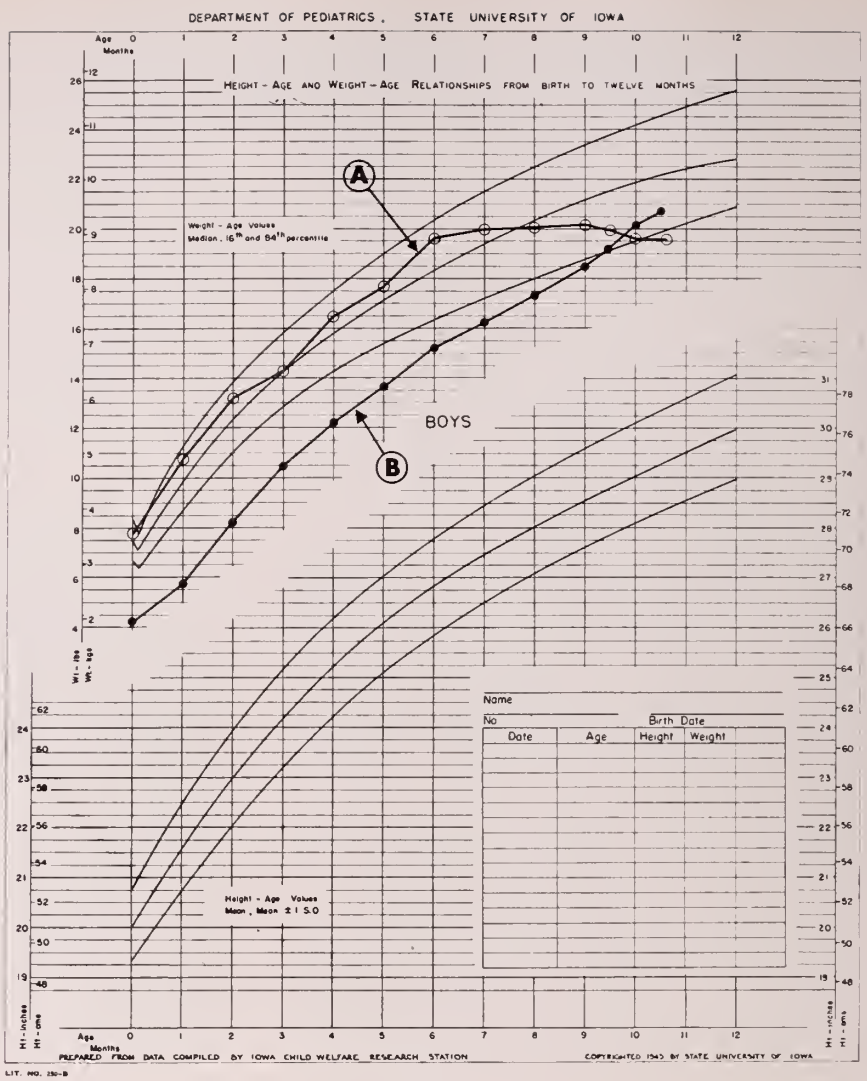


Figure 1

In addition to general body growth, it is important for the physician to be well acquainted with the various modes of growth of different organ systems. The central nervous system accomplishes fifty per cent of its postnatal growth in the first year after birth, 75 per cent by the third year, and over 90 per cent by the seventh year. Thus it is important to record head circumference measurements as an index of cerebral growth during the first few years of life, but serial measurements are not necessary routinely in later childhood because the growth of the brain is virtually complete.

Developmental behavior is equally as important as physical growth to the pediatrician. This aspect of growth and development will be covered in this Symposium.

Growth is influenced by many factors in this area. It is well known that heredity, sex, race, illegitimacy and prematurity are all important factors to be considered in assessing growth. Congenital malformation of almost any body system, malignancy, poor environmental hygiene and multiple trauma all may contribute to growth failure. Growth failure may be on a primary psychologic basis and emotional factors often contribute to growth failure by complicating the primary organic disease. In this Symposium, however, no attempt will be made to cover all facets of the problem but only certain selected aspects of physical and developmental growth.

In certain cases no demonstrable cause can be found to explain the growth failure, thus no specific treatment is possible. Unfortunately, in other cases the cause when found may not be correctable. When the

physician is satisfied that he has made a thorough examination of a child with growth failure, and that he has followed the child for a sufficiently long period of time to be certain that no etiologic factors have been overlooked, he must conclude that the child is small but otherwise healthy. His task is not finished, however, until he persuades the parents and ultimately the child to accept the fact that some human beings are small and for this there is no cure.

ENDOCRINE ASPECTS: Doctor Keele

MOST OF THE hormones have an important part in the linear growth of the young, but in contrast to widespread belief, endocrine dysfunction is not a common cause of growth retardation. Bone, nutritional, metabolic, renal and circulatory disturbances as discussed by other participants in this symposium are more common causes of growth retardation and should be considered first in the work-up of any child with this problem. However, untreated endocrine dysfunctions are associated with growth failure, and for this reason an evaluation of the linear growth becomes an important part in the diagnosis and the management of endocrine disorders.

No one hormone is solely responsible for influencing growth. Somatotrophin (growth hormone), thyroid hormone, the sex hormones, and probably insulin all participate in the growth of the child, and for this reason it is probably best to think of the hormones as being "synergestic" in their influences on growth. It is impossible at the present time to quantitate the influences of each of these on growth but that they all do influence growth has been adequately documented. This will be shown below.

There are several lines of evidence to support the role of somatotrophin in human linear growth. First, patients with organic lesions of the pituitary gland have growth *retardation;* this has been shown in patients with craniopharyngiomas,[3] Hand-Schuller-Christian Disease,[5] and possibly in toxoplasmosis.[6] However, these are usually associated with lack of other trophic hormones which may have had some casual relationship to the dwarfism. Secondly, growth hormone, extracted from the pituitary glands of man and monkeys causes linear growth in pituitary dwarfs.[1,23] Thirdly, eosinophilic adenomas of the pituitary gland cause gigantism before epiphyseal closure and acromegaly in adults.[7] Fourthly, Daughaday and co-workers[8] have shown that normal human serum contains a factor which stimulates the in vitro incorporation of sulfate-S^{35} into cartilage from hypophysectomized rats. Children and adults seem to have similar levels of this factor. The factor is almost absent from the serum of patients with pituitary dwarfism, Sheehan's syndrome (post partum necrosis to the pituitary), post-hypophysectomy lesions, and pituitary neoplasia. This factor tends to be increased in patients with active acromegaly and is normal in patients with inactive acromegaly. The sulfation factor is increased in the sera of hypopituitary patients after treatment with primate growth hormone. Although this factor may not be growth hormone, it is at least growth hormone dependent. Read[9] has obtained similar results by using an anti-human growth hormone serum produced in rabbits. That children and adults have similar levels is consistent with the observation by Gemzell and Heijenskjold[29] that growth hormone content of the pituitary glands does not change with age.

Somatotrophin probably increases human growth by protein anabolism. This has been shown to be true in animals,[10] and is associated with nitrogen retention. Somatotrophin also causes nitrogen retention in man.[1,24,12] One can speculate that somatotrophin causes human growth by accelerating chondrogenesis and bone growth just as it does in the rat.[11] There is also calcium and phosphorous retention in the treatment of pituitary dwarfs with somatotrophin.[1] Growth hormone does not cause significant bone maturation.

It is difficult to make a positive diagnosis of pituitary dwarfism in early childhood. A specific test for demonstrating growth hormone has not been available; the sulfation factor test described by Daughaday et al.[8] or the immunologic method of measuring GH

described by Read[9] may prove of value in this regard in the future. However, the diagnosis can be made with a high degree of precision if a local lesion such as a craniopharyngioma, a glioma, or Hand-Shuller-Christian Disease is demonstrated in association with dwarfism. Although such cases are rare, these lesions should be looked for by getting x-rays of the skull, especially if diabetes insipidus is present. In the majority of cases such a lesion is not present and the diagnosis is made by demonstrating the absence of the other pituitary hormones. Specific tests for the various pituitary trophic hormones are not generally available and for this reason it is necessary to look for deficiencies in the action of their target organs. Secondary hypothyroidism may or may not be present. Signs of severe hypothyroidism such as myxedema, sluggishness, constiptation are rarely seen; some may show minimal findings such as coolness and mottling of the skin and moderate lowering of the thyroid function studies. Many may have no evidence of thyroid dysfunction other than a retardation of bone age which cannot be attributed solely to thyroid hormone deficiency. Likewise, evidence of secondary hypoadrenocorticism may be absent. Occasionally one will be seen with hypoglycemic episodes and insulin sensitivity. Such findings if present are very helpful in making the diagnosis before puberty.

Of course, lack of gonadotrophic activity cannot be demonstrated until after the expected time of appearance of puberty. This can be demonstrated by the persistence of sexual infantilism and a urinary follicle stimulating hormone level of less than six m. u. per 24 hours. A positive diagnosis of pituitary dwarfism can be made if no gonadotrophic activity can be demonstrated in a dwarf with evidences of secondary hypothyroidism and hypoadrenocorticism. It is often difficult, however, to distinguish these individuals from patients with another entity called delayed puberty. In this condition the patients mature sexually and have their pubertal growth spurt two to four years late in contrast to patients with pituitary disease in which the sexual infantilism and dwarfism persists indefinitely. These patients with delayed onset of puberty also frequently give a history of lagging slightly behind their contemporaries in growth throughout childhood just as in hypopituitarism. They also tend to have a two to three year delay in bone maturation. The other parameters of pituitary function are normal. The cause is completely unknown; however, a positive family history of delayed adolescence can often be obtained, which may indicate a genetic trait.

The treatment of pituitary dwarfism presents a problem simply because there is no ideal drug commercially available. It has been well documented that primate growth hormone will result in growth of these individuals. For obvious reasons, this material is scarce, and the effectiveness of animal growth hormone in man has not been demonstrated. It is hoped that an active principle may be obtained from animal growth hormone which will be effective in man.[23]

Sex hormone treatment is indicated in proven hypopituitarism to produce a growth spurt and sexual maturity. Sometimes sex hormone treatment is indicated in delayed puberty especially when there are psychological problems relating to the sexual infantilism and delay in growth spurt. There is considerable controversy concerning the effectiveness of the androgens in children with other non-specific causes of growth failure. Sobel et al.[17] think that methyltestosterone produces more bone maturation than growth resulting in impairment of eventual stature. Bayley and co-workers[16] in testing its use in immature boys think that treatment with small doses, 10 mgm per day, produces more ultimate growth than occurs in untreated patients. They think that this dosage has the advantage of avoiding undesirable masculinization. We must leave this question open until further clinical trials have proven its effectiveness, or the effectiveness of some closely related compound.

That thyroid hormone influences linear growth is shown by the marked dwarfism in cretins and the cessation of growth in acquired hypothyroidism. Normal growth can be attained when these patients are treated with physiologic dosages of thyroid hormone. Thyroid hormone is moderate in its

bone growth promoting activity, but is very active in promoting bone maturation.[11] In the child, absence of thyroid hormone results in marked lack of growth of bone, retention of infantile skeletal proportions, and marked retardation of bone age.

The diagnosis of cretinism is difficult to make in the neonatal period. Most of these patients appear normal at birth but by three weeks of age the first symptoms usually appear and include: constipation; wrinkling of the brow; poor feeding; and cool, mottled extremities. The typical picture of cretinism with the characteristic facies of myxedema does not appear until three to six months of age at which time the chances of permanent mental damage are increased. In the absence of thyroid hormone the brain fails to grow in the newborn rat[14] and histologic damage has been demonstrated in man.[21] For this reason it is important to make the diagnosis as early as possible. In any neonate with the above symptoms retardation of bone age and lowering of the Butanol Extractable Iodine and Protein Bound Iodine should be looked for. Treatment with thyroid hormone should be started if cretinism is suspected in order to allow maximum brain growth and preserve potential mental development.[22] Various regimens are available in the literature.[7,15] Normal serum levels for BEI and PBI are also available.[15]

Children with acquired hypothyroidism usually are not so dwarfed and some may be in the normal range for height-age. These patients usually do not have permanent mental damage because the brain has finished most of its growth at the time of occurrence of the disease. A diagnosis ordinarily can be made by finding a lowered PBI, BEI, and radioactive iodine uptake. Normal mental activity and linear growth can be expected on treatment with physiologic dosages of thyroid hormone.

Untreated diabetes mellitus will result in growth retardation; rarely, however, does juvenile diabetes escape detection and treatment before marked growth failure occurs. Poorly controlled diabetes will result in dwarfism.[2,20] This seems to be related to hypersensitiveness to quick acting insulin and has not been commonly seen since the advent of the long-acting insulins. The mechanism by which insulin increases linear growth is not known. Animal experiments suggest that insulin is essential for protein-anabolism and that it is essential for the maximum anabolic response to growth hormone.[18]

Dwarfism occurs ultimately in sexual precocity. Sexual precocity is associated with excess sex hormone production. This occurs in constitutional precocious puberty, androgen producing lesions of the adrenal, and in certain gonadal tumors. Excesses of sex hormones, androgen and estrogen, in early childhood produce increased growth; however, the rate of bone maturation is increased over linear growth resulting in early closure of the epiphyses and ultimate sacrifice of adult height. Treatment to prevent the dwarfism of constitutional precocious puberty is not available. In congenital adrenal hyperplasia, early rapid growth and early fusion of epiphyses can be prevented by treating the patients with daily physiologic dosages of the glucocorticoids which inhibit the excess androgen production by the hyperplastic gland. In adrenal and gonadal tumors, surgical removal is desirable.

Dwarfism also occurs in gonadal aplasia in females, a syndrome first described by H. H. Turner,[25] and later by Varney et al.,[26] Albright et al.[27] and Wilkins.[28] These patients have, in addition to dwarfism, elevated urinary levels of gonadotrophins, sexual infantilism, and other congenital defects. About 80 per cent are sex chromotin negative (male pattern). The cause of the dwarfism is unknown. Femininization and some growth can be produced by estrogen treatment.

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METABOLIC ASPECTS: Doctor Kirkman

ONE DEFINITION of metabolism is: "the sum of all the physical and chemical processes by which living, organized substance is produced and maintained."[1] With this broad scope in mind, we shall cite not only those disorders conventionally accepted as metabolic in nature, but also those disorders not covered by speakers on other categories.

When confronted with an actual growth failure of possible metabolic origin it may be diagnostically helpful for the examining physician to think in the same sequence as occurs in the utilization of food and conversion of food to energy or tissue—from grocery store to glycogen stores.

The relative abundancy of food and the means for its purchase in this country make starvation or malnutrition an unusual—but not unseen—occurrence. In addition to economic deprivation, inadequate intake of nourishment from other causes must be considered in instances of growth failure. The question of whether a new, breast-fed infant is getting enough milk is a common one. The new mother may misunderstand instructions for formula or may fail to learn that the holes of rubber nipples may have to be widened. Over-conservatism or failure to appreciate the greater individual caloric needs of a more active infant may result in undue growth failure in an older premature infant in the nursery. A monotonous diet is as discouraging to children as to adults. Specific dietary deficiencies, such as vitamin or amino-acid deficiency are rare and overly emphasized in this country. The lay recognition of the nutritional need for milk in infancy and childhood has obviated most of the deficiencies except those of vitamin C and iron, items in which milk is deficient. Even these two deficiencies can occur with surprisingly little departure from expected weight. However, the possibility of a specific deficiency must be considered when abnormal diets are imposed on a child for reasons of allergy or absorptive problems. While the practicing physician may prescribe various vitamins in the hope of placating the parents or perhaps actually increasing the appetite of the child, he will generally be disappointed in this regard and may, in the specific case of idiopathic hypercalcemia or in the general case of detracting from the pursuit of the true diagnosis, do more harm than good.

Failure to feed due to neuromuscular, psychologic, or mental deficiencies will be discussed by others on this panel. Conditions which interfere with delivery of food to the intestines may result in growth failure and should be apparent from a history of vomiting or difficulty in feeding. These conditions include posterior-nasal atresia, rumination, esophageal anomalies, muscular disorders of gastric rhythm, pyloric stenosis, duodenal atresia, etc. A deficiency of pancreatic enzymes, as in fibrocystic disease,[2] may account for the poor digestion of an otherwise

adequate diet. Celiac disease[3] in children and older infants, and steatorrhea in the younger infants are expressions of an inadequate absorption of digested food-stuff by the intestinal wall. Both disorders are characterized by loose stools and increased concentration of unabsorbed fat in the stools. In celiac disease the oral administration of glucose or vitamin A produces less elevation of the blood-levels of these substances than in normal persons, yet the intravenous glucose tolerance curves in celiac disease may be normal.[3] These tests illustrate the defective intestinal absorption that exists in celiac disease. The various other forms of diarrhea may also cause failure in growth because of poor absorption.

Once absorbed, further actions on the intermediates may be impaired because of hepatic injury—either metabolic, as in the cirrhosis of galactosemia,[4,5] fibrocystic disease, and one of the glycogen storage diseases—or infectious. In the Von Gierke's type of glycogen storage disease, the liver lacks glucose-6-phosphatase and miserly refuses to return to the general circulation, in times of need, that glucose which it has received in times of feeding.[6] The liver enlarges from this accumulation of glucose as glycogen in Von Gierke's disease, and the administration of epinephrine fails to produce the usual elevation of blood glucose.

Growth may be impaired due to specific abnormalities of the chondro-osseous framework, as chondrodystrophy, Morquio's disease, and osteogenesis imperfecta. In this regard it is wise, of course, to search for those constellations of signs which portray the condition as a recognizable syndrome or disorder—as the characteristic disproportions of the chondrodystrophic dwarf, or the features of gargoylism.[7] In the latter instance, and in cases of renal disease, idiopathic hypercalcemia, lipidoses,[8] and untreated galactosemia, the chemical manifestations are becoming known, but the exact ways in which synthesis of tissue is impaired are not known. But for the examining physician, the problem of diagnosis of metabolic growth failure lies not among such physically apparent disorders as chondrodystrophy, Morquio's, celiac, glycogen storage, Niemann-Pick disease, etc; the relatively striking physical findings in these instances will cause the examiner to make the diagnosis or consult the literature pertinent to the appropriate physical signs. Indeed, some of these disorders are not primarily of concern as growth problems. The concern is rather with the disorders with the more elusive or misleading findings. Notoriously belonging to this group are fibrocystic disease,[2] idiopathic hypercalcemia,[9] galactosemia,[4,5] and the renal tubular defects.[10] It behooves us, therefore, to become familiar with the suggestive symptoms of these disorders and the laboratory tests appropriate to their diagnosis.

The victims of fibrocystic disease, synonymously known as cystic fibrosis of the pancreas or mucoviscidosis secrete a mucin of high viscosity and a sweat of high concentration in electrolytes. As a consequence of the abnormal mucin, obstructions of the pancreas and of the broncial and biliary trees may occur. Meconium ileus, impaired digestion of proteins, and chronic respiratory distress or infection may result. The diagnosis of fibrocystic disease is confirmed by the finding of a deficiency of duodenal trypsin or an excessive concentration of chloride in sweat. Children with the milder forms of idiopathic hypercalcemia may exhibit only anorexia, irritability, and some vomiting. Listlessness and obvious growth failure follow. Elevation of serum calcium and moderate albuminuria occur. Young infants with untreated galactosemia have hepatomegaly, vomiting, severe growth failure, and often jaundice. If they do not die, they lose their jaundice but often develop cirrhosis, cataracts, and mental deficiency. The excretion of urinary substance (galactose) which reduces Benedict's solution but fails to reduce glucose oxidose test paper, is characteristic of the galactosemic child taking milk. Highly reliable enzymic blood tests exist for the diagnosis of galactosemia. Victims of the renal tubular defects may have rickets or merely non-specific symptoms accompanying growth failure. Their diagnosis is suspected by the findings on routine urinalysis, combined with determinations of serum electrolytes and bicarbonate. Urinary amino-acid determinations may be confirmatory.

Recent, very powerful tools in biochemical

investigations have clarified some of these metabolic problems and promise to unveil new causes for growth failure. But, in spite of these technical tools the fact remains that all of these presently-known metabolic disorders are tentatively diagnosable by a well-taken history and physical examination and by a selected group of more or less routine laboratory tests. From this tentative evidence the more specific or confirmatory tests may be chosen.

These metabolic diseases are more than just biochemical oddities of interest only to a small group of academic investigators. Diagnosis of these diseases provides very definite information for prognostic and genetic counseling. Early detection and treatment of such disorders as celiac disease and galactosemia may be life-saving and may permit the individual to lead a normal life. Appropriate treatment can permit the victims of fibrocystic disease, glycogen storage disease, and renal tubular disorders to lead longer and more comfortable lives. These measures can be taken with a clear conscience toward possible pollution of the human genetic stock. Most of these disorders are relatively uncommon, recessive diseases. This means that symptomless carriers of the disease, rather than the affected individual, are responsible for the perpetuation of the mutations. Indeed, by his recognition of the disorder and genetic counseling to the parents, the practicing physician has contributed his bit toward the genetic hygiene of his species.

In conclusion, metabolic disturbances often express themselves in infancy and childhood as growth failure. To the practitioner, this is both a helpful diagnostic sign and an alarm as to urgency of needed action, for metabolic causes of this failure may, if untreated, deprive children of their rightful legacy of life and growth.

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NEUROLOGIC ASPECTS: Doctor Waites

AN UNDETERMINED NUMBER of infants who do not thrive have cerebral dysgenesis (or agenesis) as the basic pathological process in their difficulty. This may be manifest in a variety of clinical forms. Dysgenesis implies that the nerve cells of the brain do not develop as normal neuroblasts, or that defective maturation interposed between the neuroblast stage and the mature neuron. Either abnormal neuroblasts or defective maturation would subsequently result in neuronal degeneration and the clinical manifestations of cerebral dysgenesis. It naturally follows that the white matter of the nervous system will not be normal if the grey matter with its nerve processes does not progress through the usual embryological stages to maturity. These conditions have been adequately demonstrated pathologically from the most severe microcephaly, with no differentiation between grey and white matter, to the most discrete nerve involvement of a single functional system of the brain.

For this discussion, we will consider the conditions that have been termed historically "cerebral spastic paralysis," "Little's disease," "double infantile spastic hemiplegia," "spastic diplegia," "spastic paraplegia," and now have the glorious misnomer of "cerebral palsy."

Little published his famous monograph in 1861 and first called attention to "the influence of abnormal parturition, difficult labor, premature birth, and asphyxia neonatorum, on the mental and physical condition of the child, especially in relation to deformities." His clinical descriptions, observations, and deductions therefrom are legendary and have hardly been improved upon to the present. He did not believe gross injury to the brain to be the causative factor, and he did not believe meningeal hemorrhage to be a factor. He did establish the fact that the condition is often associat-

ed with abnormalities of birth, but he also states that many infants born of difficult labor and delivery would subsequently be normal. It was his opinion that the pathology was a widely spread fine lesion of the cerebral substance, and that it may affect one part of the brain more than another. He reasoned that the most common causal factor was asphyxia.

In 1885, Sara McNutt presented a thesis to the American Neurological Association concerning this same subject. This particular thesis is of much significance to us today. The clinical descriptions and pathological findings in this paper are excellent, but the erroneous deductions from these findings have remained with us to this day. She reasoned that the entire process of double infantile spastic hemiplegia was produced by meningeal hemorrhage in her cases, and consequently, hemorrhage was the etiological factor in infantile spastic states dating from birth and associated with difficult labor. This erroneous deduction has perpetuated the most amazing and unbelievable influence upon medical thought concerning spastic and related states, and only recently have other concepts gained general medical credence. Even today it is not uncommon to hear that an infant has difficulty because of "birth trauma and/or hemorrhage," whereas neither of these morbid conditions can honestly be incriminated.

In 1897, Freud summarized some of his observations in the monograph "Infantile Cerebral Paralysis." He included a critical analysis of all previous papers on this subject. This is still the most complete essay on the subject. He was the first to use the term "cerebral diplegia" in reference to bilateral symmetrical defects in the brain, not related to gross lesions. The result of gross lesions he termed hemiplegia and double hemiplegia. He proved that diplegia had its pathological origin long before birth in intra-uterine existence, and largely disproved that meningeal hemorrhage was any factor in diplegia or hemiplegia. Freud summarizes: "Premature, precipitate and difficult birth, and asphyxia neonatorum are not causal factors in the production of diplegia; they are only associated symptoms of deeper lying influences which have dominated the development of the foetus or the organisms of the mother." He was of the opinion that the cerebral paralysis results from difficult births when the brain is lacerated or contused, and that this is manifest as a monoplegia or hemiplegia, very rarely as a double hemiplegia. The anatomical changes in the brain would then be entirely different from those of diplegia. After disproving the traumatic delivery theory as a causative factor, Freud connected the prenatally developed diplegia with those cases in which the onset may occur at any age from birth even to puberty. He argued strongly for a primary neuronal degeneration of the central nervous system which might become manifest during any phase of fetal life or childhood.

In 1909, Anglane and Jacquin reported the findings in a patient with spastic diplegia in which there was neuronal degeneration in the cerebellum, as well as extensive neuronal degeneration in the cerebrum. This may be the first report of such involvement in the cerebellum. Batten, an English neurologist with more than usual interest in pediatric neurology, postulated that the cerebellum could be involved in some types of spastic disease. He believed that the cerebellum was small because of neuronal involvement, and not because of lack of the cerebro-ponto-cerebellar fibres as had been postulated by some of his contemporaries.

Collier, in 1924, again suggested that "agenesia" is a primary neuronal defect, and is responsible for essentially all cases of diplegia. His article in *Brain*, 1924, is well worth one's attention and is the primary source for these remarks.

Recent workers from the Continent have suggested that venous congestion may be an important factor in spastic states, and related conditions. Their attention has been focused on the area of the great vein of Galen and its tributaries—much investigation remains to be completed before their final answer will be forthcoming.

We may someday realize the importance of the embryology of the specific functional systems of the brain and the temporal relationship of one system to the other. The problem then resolves itself to determining the etiology of neuronal involvement in each

system that may show pathological development, and the subsequent clinical manifestations.

From the work that has been done in this field to the present time, it is safe to assume that most of the infants that fail to thrive because of central nervous system involvement had this difficulty before they were born. The clinical picture is a result of the prenatal defect. Birth trauma, hemorrhage, asphyxia, anoxia, and other such morbid factors may well contribute to the difficulty, but are not the etiology.

When the patho-physiology of cerebral dysgenesis is completely understood, it is likely that the pathogenesis of the more subtle neurological disturbances in infancy will be quite clear.

PSYCHOLOGICAL, SENSORY and PERCEPTUAL ASPECTS: Doctor Richardson

COMPARATIVELY LITTLE is known about normal psychologic, sensory and perceptual growth and development, especially in infancy. Our increasing awareness of the pathology in these areas, however, is stimulating quite a bit of research, the results of which promise to be rewarding.

The earliest sign of psychologic, sensory or perceptual dysfunction is most frequently seen in the delayed or faulty acquisition of speech or language, the latter being that symbolic function which includes all forms of communication.

Best and Taylor describe normal speech development (including the symbolic function of language): "The first stage in the development of speech is the association of certain sounds (words) with visual, tactile and other sensations aroused by objects in the external world. These associations are 'stored' as memories. After definite meanings are attached to certain words, pathways between the auditory area of the cortex and the motor area for the muscles of articulation become established, and the child attempts to formulate and to pronounce the words which he has heard. This act of verbal expression involves the coordinated movements of a large group of respiratory, laryngeal, lingual, pharyngeal and labial muscles. Later, as the child is taught to read, auditory speech is associated with the visual symbols of speech and, finally, through an association between these and the motor area for the hand, the child learns to express his auditory and visual impressions by the written word." It has been stated truly that speech is the greatest interest and the most distinctive achievement of man!

Language development has both physical and psychological components. It requires adequate integrity of the peripheral nervous system (sensory capacities), of the central nervous system, and adequate integrity psychologically. Anatomical integrity of the articulary apparatus is necessary of course.

We might review briefly the normal sequence of speech development. The child begins to use single words to express his needs usually between ten months and two and one-half years of age. Two or three word phrases are introduced between eighteen months and three and one-half years, and an ever increasing vocabulary follows. Although the period of speech development may normally extend from nine months to three and one-half years, the period between one and two years of age is in most children that of maximum rate of speech development, and after this period speech is acquired with less facility.

It has always been recognized that retarded mental development is closely allied with delay in the utilization of speech skills as well as in decreased language content. Mental retardation interferes with full comprehension, although all but the most severe who have normal hearing understand simple speech associated with the basic needs of life.

If hearing is impaired, the child may fail completely to speak, or language development may be retarded and articulation defective. The intelligent hard of hearing child may show an interest in oral communication and may attempt to compensate for his hearing disability through lip reading. If the hearing loss is severe and the child is able to hear only noises he may be certified erroneously as mentally defective. General response to sound in such a child might be regarded as an indication of normal hear-

ing, and failure to understand fully what is said, or to make suitable progress in school, might be taken as a sign of retardation. Inasmuch as speech is learned chiefly through the hearing mechanism by imitation, the child learns to speak not only *because* he hears, but *as* he hears.

If development of speech is delayed well beyond the range of normal, and the child's ability to formulate his thoughts into language is grossly inadequate for his age, the condition may be described as a developmental dysphasia or a central communication disorder. Such a disorder may be associated with spastic and related conditions, or may exist in the absence of any obvious underlying neurologic disorder. It may be the result of some cerebral dysgenesis producing delay in the use of the symbols of speech. The child with delayed neurologic maturation of the central processes involved in the use of expressive speech may have adequate understanding of language. He may be friendly and cooperative in his attempts to communicate with others and yet be unable to express himself through speech. He may appear to have little or no interest in the imitation of sounds, having been a silent child who made little attempt to vocalize or to babble during infancy. He may show signs of frustration when he cannot make known his meaning and may rely chiefly on gesture or well-developed mime to achieve his essential need for communication. This has been called expressive or motor dysphasia.

In other instances there may be lacking or limited appreciation for the meaning of spoken language which is not related either to peripheral auditory impairment or subnormal intelligence. This condition has been described as receptive dysphasia or congenital auditory imperception. These children may also use gesture to supplement their limited means of verbal expression. They generally show little interest in speech as a means of communication, often fail to attempt speech, and make little or no attempt to use the visual sensory pathways such as lip reading. The child who has no understanding of language and who cannot express himself orally is said to have a mixed type of dysphasia. It is actually doubtful that there is a pure form of dysphasia at all.

There are as yet relatively few studies on perceptual deviations as such. However, the studies which have been done on children with learning disabilities such as specific reading disability, or dyslexia, have led some of us to believe that perceptual disorders are not uncommon in children with delayed language development. Though intelligence depends on the functioning of many parts of the brain, certain areas have been located that investigators believe are more particularly associated with perceptive processes. Association paths between these areas are known to exist, and there are always nerves which relay impressions from the sense organs to the brain centers, or from the brain to motor and sensory mechanisms. Many combinations of injury or faulty development are therefore possible, and the potential effects on the child's perceptive and thought processes are legion. Also a sensory defect interferes with perception in the range of action of the defective sense. Therefore, if there are also sensory handicaps, their exact nature and their effect on perception need to be carefully determined. It must also be pointed out that perceptive or thought defects can be caused by emotional problems or personality distortion.

Strauss defines perception as intermediate between sensation and thought, calling it "the mental process which gives particular meaning and significance to a given sensation and therefore acts as the preliminary to thinking. It is the means by which the individual organizes and comes to understand the phenomena which constantly impinge upon him." He describes two particular aspects of perception as follows: (1) Perception, in terms of Gestalt psychology, is a psychologic process in which each part of a whole is seen or heard or felt in relationship to the other parts, resulting in a figure which is recognized immediately and uniquely. (2) Another characteristic of normal perception is the fact that a whole is perceived as a foreground figure against a background. We have seen how some children with central nervous system dysfunction or dysgenesis are unable to coordinate parts of a simple whole, like the sides of a triangle,

into a correctly integrated form. In addition, when figures of test material are presented against a background, these children cannot differentiate the foreground from the background. Moreover, in trying to assemble test material which involves the use of motor, visual, or hearing abilities, some children may be incoherent and disorganized in their attempts. Many now feel that children with specific reading disability or other communication disorders have this type of visual-motor perceptual difficulty. Perceptual deviations have been commonly attributed to specific neural lesions, but they seem similar to performance patterns which are normally expected of children at lower levels of perceptual maturation; therefore, the possibility of retarded or arrested maturation should be considered in evaluating what are apparently perceptual defects.

Interference with the development of speech can occur if emotional growth is not progressing harmoniously. There may be little need or desire for contact or communication with others. Speech, once acquired, may not be used freely or may be almost entirely inhibited. Extreme shyness or self-consciousness can hinder the gradual development and stabilization of speech. If the arrested or defective development of speech is the result of, or associated with, true psychogenic retardation, there will probably be signs of emotional difficulties and maladjustment in other respects. It is not uncommon, however, for the emotional disturbance to be secondary to the speech disorder, especially where much frustration has been experienced by a child with developmental dysphasia or unintelligible articulation. Many children refuse to speak and take refuge in silence when they realize their inability to communicate effectively, or when they are subjected to repeated and misdirected correction of their speech at home.

There are many so-called functional or environmental causes for delayed speech. These would include prolonged or serious illness during the first year of life which interferes with the baby's vocal play and prelanguage activity; over-protective or rejecting parents; poor speech models in the home; the lack of a mother or a mother-figure in close relationship with the child; over-anxious parents who try to force or cajole the child into speaking; etc.

The delayed establishment of cerebral dominance used to be considered an etiological factor in stuttering. This has not been borne out, but there is strong evidence that delayed development of cerebral dominance may be causal in delayed speech. Dominance develops gradually and is normally established between the age of three and six years. Nielsen points out that many children show definite laterality at one year. Speech readiness usually develops along with dominance and if the latter is delayed, the former is usually similarly retarded. Evidence has seemed convincing that there is a higher incidence of speech disorders, learning disabilities, and behavior disorders in children whose dominance is not "set," that is, who have a lack of unilateral hand preference, than in those who are strongly right or left handed. However, there is lack of agreement as to whether a cause and effect relationship exists or whether these conditions simply occur concurrently. Cerebral dominance is said to involve not only hand dominance but also eye dominance. Berner and Berner concluded from the study of 500 cases with and without reading difficulties, defects of speech, and allied visual-motor disorganization, that unless the controlling eye in binocular vision is on the side of the dominant hand, a chain of symptoms referable to eye-hand confusion is likely to occur. Further study is needed to throw light on these controversial issues and help to increase understanding of what disturbed dominance means.

In summary, the delayed or faulty acquisition of speech or language may be one of the earliest and most signifcant signs of psychological, sensory or perceptual dysfunction. Faced with such a child, we have to decide whether this is due to mental deficiency, severe or partial hearing loss, to developmental dysphasia or to psychogenic factors. There are numerous environmental factors which may also be etiologic. The relationship between cerebral dominance and communication skills has yet to be clarified.

CARDIAC ASPECTS: Doctor Cayler

THERE IS an abundance of clinical infortion concerning growth failure or retardation in children with congenital heart disease, but the basic mechanisms of growth failure in these patients is not well understood. Everyone working in the field has opinions based on clinical observations. There are several reports of clinical studies documenting abnormal growth patterns and changes in growth patterns after surgical therapy. There is, however, very little information concerning the actual etiology of growth failure in these patients.

One bottle-neck to etiologic investigations has been the lack of information concerning normal hemodynamics in infants and children. Without normal data it is impossible to intelligently interpret the significance of abnormal data from patients with heart defects. The reasons for the pausity of normal data is due to the fact that no easy clinical method is available for obtaining detailed accurate hemodynamic information. At present the cardio-pulmonary laboratory is the only place where these type studies can be performed. Investigators are justifiably hesitant about subjecting normal infants to the type studies needed. Another source of difficulty is in actually obtaining enough voluntary, or involuntary in the case of infants, cooperation to allow controlled hemodynamic measurements.

The circulatory system of course is vital to all the body functions including growth. It normally carries oxygen and nutrients to the capillaries and maintains an adequate diffusion gradient to allow exchange of gases and metabolites between the vascular system and the tissues. Congenital heart disease is generally considered to interfere with normal tissue metabolism and growth by resulting in:

1) a decrease in systemic arterial oxygen saturation (right to left shunt) or 2) a decrease in systemic cardiac output. Although in general the degree of growth retardation correlates with the severity of the heart defect, exceptions are not uncommon both in terms of marked growth failure with minimal heart disease and minimal or no growth failure with very severe heart disease.

Growth failure due to systemic arterial unsaturation and decreased tissue oxygen delivery is easy to appreciate. When severe, even the compensatory mechanisms of polycythemia, changes in the hemoglobin-oxygen dissociation curve and perhaps increased cardiac output do not allow adequate basal or resting oxygen delivery. Some observers have reported an apparent compensatory decrease in oxygen utilization or a decreased basal metabolic rate in these patients,[1] but this has not been found by others.[2] Growth failure may not be a good term for patients with cyanotic heart disease. Growth retardation is more appropriate since the majority of these patients eventually attain full physical development.[3] It is of interest that the growth of patients with cyanotic heart disease is occasionally actually greater than normal. This is seen most commonly in patients with isolated pulmonary stenosis. It is possible that the explanation for this is related to the increased caloric intake and limited energy expenditure (e.g. marked physical inactivity) which is often seen in these patients.

It is also easy to understand how an actual decrease in basal or resting cardiac output may interfere with tissue metabolism and growth. Such a decrease has been demonstrated in adults with congenital heart disease but has not been found in children.[4] Until recently no output studies had been performed on infants with congenital heart disease. Table one lists the data on systemic cardiac output for a group of patients with congenital heart disease whose ages range from ten weeks to forty years.[2] The difference in resting cardiac output between the older groups is apparent. No difference in resting cardiac output was demonstrated however between the control (class 1) or diseased (classes 2, 3, 4) infants or smaller children, even though growth retardation in these later patients was usually quite marked. It is possible that the difference between the cardiac output necessary to sustain life and the cardiac output necessary to permit normal physical development is so small in infants that our relatively crude methods of measurement are inadequate to demonstrate the difference.

RESTING CARDIAC INDEX

	B.S.A. (m^2)	AGE	SEVERITY OF HEART LESION	CARDIAC INDEX (L/Min)
1.	0.2-0.5	<2 yrs.	1. Class 1	—
			2. Class 2	5.0
			3. Class 3 & 4	5.0
2.	0.5-1.0	5-10	1. Class 1	5.0
			2. Class 2	5.0
			3. Class 3 & 4	5.0
3.	1.0-1.5	10-15	1. Class 1	5.0
			2. Class 2	4.0
			3. Class 3 & 4	3.0
4.	1.5 +	15+	1. Class 1	4.5
			2. Class 2	3.5
			3. Class 3 & 4	3.0

TABLE ONE

The effect of congenital heart disease on resting systemic cardiac output in infants and children.

The pattern of growth failure due to congenital heart disease is one of greater interference with weight growth than length or height growth.[5] This is best demonstrated on an anthropometric chart where one almost always observes that the weight percentile is significantly lower than the length or height percentile in these patients. If this pattern of growth failure is not seen it is most likely that the growth failure is not due to heart disease.

Physical growth failure due to congenital heart disease is not static. Growth retardation is usually not progressively more severe after infancy. Although growth usually continues at about the same rate after infancy, appreciable spontaneous improved growth is not infrequently seen. In a series of infants and children with ventricular septal defects, nine of thirty-two (28 per cent) showed improved growth after the age of one year.[6] Engle[7] and others[8,9] have shown that surgical correction of patent ductus arteriosus in children is often not accompanied by improved growth. Present data suggests that the earlier the surgical correction is performed in relation to the onset of growth failure, the greater is the chance of obtaining normal growth.

Although puberty may be delayed in the child with severe congenital heart disease, there is no good evidence to indicate that the heart disease results in significant secondary endocrine abnormalities which may cause growth failure. Rarely a child may have both congenital heart disease and an endocrine deficiency. Correction of the heart disease does not cure the endocrine problem.

In conclusion, it is well to emphasize that there are often primary or associated non cardiac reasons for growth failure in children with congenital heart disease. These include familial (or hereditary), emotional, nutritional, metabolic, and infectious conditions.

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INFECTIOUS ASPECTS: Doctor Riley

CHRONIC or repeated infection is probably the most frequent cause of malnutrition and growth failure in American children and a careful search for infection is indicated in every child with growth failure. As a rule, infection usually produces a decrease in appetite and interferes with proper digestion and absorption of food stuffs. Fever and other influences of infection often increase metabolic need for nutrients. Furthermore, depression of the bone marrow with resulting anemia and oxygen lack to tissue may occur as a result of infection. The inactivity which often accompanies illness may create negative nitrogen and calcium balances in a child and thereby interfere with growth. The effect of infection on growth may be either local or general. Apart from the obvious effects of anorexia and fever, however, the deceleration or arrest in growth that occurs with infection is unexplained. Gardiner-Hill[1] states that certain infections may cause a premature "senescence of growing cartilage" with calcifica-

tion. In the case of acute infection, there appears to be a compensatory mechanism which permits growth to resume its intrinsic pattern during the recovery phase. However, if infections are chronic or recurrent, even though relatively minor, growth may be significantly affected.

The most extreme examples of growth failure due to infection are seen in regions where parasitic diseases such as malaria, hookworm and tapeworm infestations occur. It has been shown that experimental animals that are rendered anemic and hypoproteinemic will utilize dietary protein preferentially for the formation of hemoglobin rather than for reconstruction of body mass and presumably skeletal development.

A study was carried out by the Social Medicine Unit of Oxford University concerning the effect of common childhood diseases on skeletal development. This study involved 650 individual children during more than 2000 child years of observation. These children experienced measles, chicken pox, scarlet fever, pertussis, respiratory, skin and intestinal infections as well as other common childhood diseases. The final statistical evaluation revealed that there was a stunting effect when the heights of a group of children who had been ill in any given time were compared with a similar group who had remained healthy.[2]

Chronic or recurrent infection of virtually any system may result in growth retardation.

Respiratory Infection:

It is well known that children with recurrent respiratory infections such as acute tonsillitis and otitis media may exhibit growth failure if there is a frequent occurrence of these infections even though the individual infection may not be clinically severe. The failure in growth of children with chronic pulmonary infections including bronchiectasis, cystic fibrosis of the pancreas, tuberculosis and pulmonary mycoses is well documented. Children with bronchial asthma are prone to develop pulmonary infections and may exhibit growth failure on this basis.

Urinary Tract Infection:

Pyelonephritis and congenital anomalies of the urinary tract should be searched for in any child who has growth failure unexplained by other causes. West and Smith[3] investigated the effects of renal insufficiency on growth. Virtually all of their patients who showed marked disturbance of growth had either bilateral involvement such as renal hypoplasia or bilateral obstructive phenomena with superimposed infection. The vast majority of patients with growth failure of renal etiology exhibit chronic renal acidosis. It is likely that the decreased food intake, due to the need to ingest large quantities of water leaving little time or gastric space available for food intake, may explain in part the resulting growth failure in such patients.

Gastrointestinal Infections:

It is also well known that infants and children with chronic or recurrent bacillary dysentery or parasitic infestations of the intestinal tract will exhibit growth failure. The constant loss of minerals and other nutrients in the stools of these patients may contribute to the growth retardation.

Infections of the Osseous System:

While local infection can increase bone length by metaphyseal revascularization, chronic osteomyelitis usually results in generalized growth retardation. Destructive vertebral lesions with secondary collapse and wedging can reduce height.

There are of course many other types of chronic or recurrent infection that could be mentioned. It is safe to say that chronic or recurrent infection in any system can result in growth retardation in certain children. It is also important that certain fundamental defects such as agammaglobulinemia which may predispose to infection in any one or several body systems not be overlooked.

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Harris D. Riley, Jr., M.D.
800 N.E. 13th, Oklahoma City, Oklahoma

Defects in the chest wall permitting herniation of the lung may arise from a variety of causes. Good repair can easily be obtained using local chest wall tissue.

THORACIC LUNG HERNIA

IRA ROSS GRIMES, M.D.

WARREN L. FELTON, II, M.D.

Ira Ross Grimes, M.D., graduated from the University of Oklahoma School of Medicine in 1954 where he is now a resident in surgery.

Since graduating from Washington University School of Medicine in 1949, Warren L. Felton II, M.D., has been certified by the American Board of Surgery and the Board of Thoracic Surgery.

Limiting his private practice to thoracic and cardiovascular surgery, Doctor Felton is also Clinical Assistant in Surgery at the University of Oklahoma School of Medicine and attending physician in Thoracic Surgery at the Veterans Administration Hospital in Oklahoma City. He is a member of the American Trudeau Society.

LUNG HERNIA may be defined as a condition in which a portion of the lung protrudes beyond the normal confines of the thoracic cavity. The herniated lung may or may not be contained in a sac of parietal pleura and is characteristically reduced at rest and herniated only on coughing, straining or expiration against the closed glottis.

Such hernias may be classified as thoracic, cervical, mediastinal or diaphragmatic according to their location. Etiologically they may be congenital or acquired, the latter being traumatic (accidental or surgical), spontaneous or pathologic.

A case of acquired, traumatic thoracic lung hernia, associated with underlying bronchiectasis, chronic lung abscess and broncho-cutaneous fistula, is presented.

Case Report

R. O. McD., a 31 year old white male construction worker, first became ill with chills, fever and productive cough in early February 1958. He was hospitalized first in his community hospital and then transferred to the Veterans Administration Hospital, Amarillo, Texas, in early March 1958. Chest roentgenograms revealed a large cavitary lesion in the posterior portion of the left lower lobe. A course of antibiotics was given with some improvement.

From the Department of Surgery of the University of Oklahoma Medical Center and the Thoracic Surgical Service of the Veterans Administration Hospital, Oklahoma City, Oklahoma.



Figure 1. Pre-operative view of patient showing the left posterior chest wall defect during normal respiration.

On March 12, 1958, he was transferred to the Veterans Administration Hospital, Oklahoma City, with the diagnosis of lung abscess of the left lower lobe and empyema. A left thoracentesis was performed shortly after admission, and 160 cc. of thick green pus was obtained which on culture produced *Klebsiella pneumonia*. He was given chloroamphenicol, and on March 17, 1958 a closed thoracotomy (tube) drainage was carried out on the left. On April 9, 1958 a large pleural air leak was noted indicating a bronchopleural fistula. On April 17, 1958 open thoracotomy drainage was performed with resection of approximately 8 cm. of the eighth and ninth left ribs posteriorly. A thick-walled empyema cavity was encountered which communicated with an abscess in the superior segment of the left lower lobe. The cavity was packed, and resolution of the purulent infection occurred. The patient was discharged from the hospital on May 5, 1958.

He did not return until his readmission on September 18, 1959, seventeen months later. He stated that he had been relatively well in the interval, but that he was troubled with the chest wall defect through which his lung bulged on coughing and which required a dressing because of the slight to moderate constant drainage. He denied cough, sputum, fever, pain or dyspnea.

Physical examination revealed a healthy appearing adult male in no distress and with normal vital signs. No abnormalities were noted except for a defect 6 cm. in diameter in the left posterior chest wall (figure 1). On coughing lung tissue with a rather glistening surface would herniate through the defect with the escape of a small amount of air (figure 2). There was no evidence of local purulent infection, and the lungs were otherwise normal to examination.

A complete blood count and urinalysis were normal. Cultures of the chest wall lesion grew *Proteus mirabilis*. Standard chest roentgenograms showed no abnormality of the lung fields except for the rib defects on the left. Bronshography on September 24, 1959 revealed contraction of the left lower lobe basilar bronchi with spillage of the contrast medium into the defect via the bronchi of the superior segment of the left lower lobe (figure 4). The remainder of the bronchial tree was normal.

On September 28, 1959 left thoracotomy was performed. The remainder of the eighth



Figure 2. Pre-operative view of patient showing lung herniating through the chest wall defect on coughing.



Figure 3. Post-operative view of patient showing the thoracotomy incision following left lower lobectomy and closure of the chest wall defect.



Figure 4. Pre-operative bronchogram (oblique view) showing moderate bronchiectasis of the left lower lobe basilar segments and filling of the peripheral bronchi out to the lung surface at the site of the chest wall defect.

rib was resected. The lining of the defect was excised, and the pleura at this site was freed of adhesions where it was continuous with the superior segment of the left lower lobe. Left lower lobectomy was carried out. The thoracotomy was closed by mobilizing the soft tissue and adjacent ribs above and below the defect. A satisfactory stable closure was obtained (figure 3). Pneumoperitoneum was induced in order to elevate the diaphragm and reduce the size of the pleural cavity temporarily.

The lower lobe specimen showed moderate basilar bronchiectasis. The lung tissue at the site of the hernia was covered by both granulation tissue and bronchial-type epithelium, and two small bronchial communications could be demonstrated. This represented then a chronic lung abscess of the superior segment with broncho-cutaneous fistulization.

The patient recovered without incident, the wound healed primarily (figure 3) and the remaining left upper lobe expanded well to fill the left hemithorax (figure 5).

Discussion

Lung herniae are not common, and the majority of those of clinical significance are acquired and thoracic in location.

Of the others, the cervical herniae are in general congenital and of limited significance. Mediastinal herniation occurs commonly in several situations (such as resection of the opposite lung), but is termed a hernia for purposes of classification only and is ordinarily of secondary clinical importance. Because of the pressure relationships herniation of the lung through a diaphragmatic defect is unlikely, and only one such possible case has been reported.[2] For further description of this group of lesions several general discussions and reviews are available.[3,4,5,6,10]

Thoracic lung hernia represents nothing more or less than a defect in the chest wall of sufficient size and weakness to permit a portion of the lung to protrude when the intrathoracic pressure is increased. Such a defect may occur as a result of infection (tuberculosis, non-specific) or malignancy. Rarely such defects develop spontaneously,[4] usually in the anterior chest near the sternum where the muscular protection of the



Figure 5. Chest roentgenogram taken two weeks post-operatively showing good expansion of the remaining left upper lobe. Residual pneumoperitoneum induced at the time of surgery can be seen.

thorax is the thinnest. Most commonly, however, thoracic lung herniae are secondary to trauma. Those that develop late after injury or operation have been termed consecutive.

Such diverse accidental injuries as stab wounds,[8] blunt injuries causing rib fractures,[13] gunshot and missile wounds, and goring by a bull[3] have led to defects permitting lung herniation. Intercostal herniation at the site of surgical thoracotomy closure occurs,[1] and it is surprising that it is not seen more often in view of the long incision with potential areas of separation. Surgical resection of rib segments for malignant disease[10] or for purposes of open drainage of the pleural space (authors' case) may, of course, leave such defects.

Operative procedures that require resection of a portion of the chest wall would more frequently result in thoracic hernia were not measures taken to re-establish the thoracic integrity at the time of the primary operation. In this regard the technical considerations in chest wall reconstruction are the same whether the lesion is an established lung hernia or a surgically or accidentally produced defect being treated primarily.

Maurer and Blades,[7] who have presented the largest single series of surgically repaired lung herniae, have emphasized the use of local chest wall tissue in their reconstruction. This includes rib, periosteum and muscle. They stress the importance of closing the defect with bone or periosteum which will produce bone. Such local tissue, with appropriate mobilization, will usually suffice to close the smaller defects (as in the present case) and in those larger ones where the long axis of the defect parallels the ribs.

Where the defect is large or of awkward shape extra-thoracic tissue or material may be required for closure. This may be necessary following the resection of costal cartilages in the extended radical operation for breast carcinoma described by Urban,[11] resection of segments of multiple ribs for chest wall tumors and in the management of close proximity missile wounds of the thorax where wide areas of the chest wall are destroyed.

Fascia lata[11,13] and tantalum mesh[1,9] have been used to provide integrity and stability to the thorax in these situations. A new material which appears most promising for this purpose is Marlex mesh.[12] This plastic mesh is easily sterilized and handled, biologically inert and of satisfactory body and strength. A major advantage over other prosthetic materials is that it apparently does not augment infection or retard wound healing even in the presence of pre-existing though properly drained infection. It is recommended that the coarser and stiffer material be used for thoracic defects and be sutured with stainless steel sutures under moderate tension, thus providing a rather stable chest wall immediately.

Perhaps the most important feature in the management of a lung hernia is the careful evaluation of the underlying lung and pleura. Bronschoscopy, bronchography, sinus tract injection, sputum studies as well as standard roentgenograms of the chest may be required for this. Surgical attack on the chest wall defect should not be undertaken without a concomitant plan of therapy

for the underlying specific or structural disease if present. In the case reported here the appropriate resectional therapy (left lower lobectomy) was carried out for the demonstrated bronchiectasis, chronic lung abscess and broncho-cutaneous fistula. The surgical management of the chest wall defect became almost incidental, despite the fact that the presence of the defect was the patient's presenting complaint.

Summary

A patient with thoracic lung hernia secondary to rib resections and with associated bronchiectasis, chronic lung abscess and broncho-cutaneous fistula is presented.

The relationship of thoracic lung hernia to hernia of the lung in general and to other chest wall defects is briefly discussed.

The use of local thoracic bone, periosteum and muscle for surgical closure of these defects where possible is stressed, and the use of prosthetic material in the larger defects is described.

Careful evaluation and management of any underlying disease of the lung and pleura is essential in the rational approach to the problem.

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Warren L. Felton II, M.D.
1200 N. Walker, Oklahoma City, Oklahoma

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Hypothermic Total Body Cardiopulmonary Bypass, Report of 19 Consecutive Cases*

NAZIH ZUHDI, M.D. JOHN MONTROY, M.D.
ALLEN GREER, M.D. JOHN CAREY, M.D.
BILL McCOLLOUGH, M.D.**

WITH THE ADVENT of open heart surgery, divergent schools of procedure have emerged. We have the advocators of normothermic perfusion and the proponents of hypothermic extracorporeal circulation.[1,2,3] Some defend a bubbler and others a filmer. Some recommend the DeBakey and others the Sigmamotor pump. Potassium citrate, acetylcholine, potassium, magnesium and neostigmine, cold, electrical current, and anoxia have been favored to obtain an empty "motionless" heart and to prevent air embolism. Retrograde perfusion of the coronary sinus and direct perfusion of the coronary arteries, with and without cardioplegia, have been tried.

It is apparent that there is ample room for confounding variants. We are not proposing hypothermic perfusion and simple occlusion of the aorta as the final answers to open heart surgery. We are merely presenting a few facts that impressed us favorably during their successful clinical application.[3,4,5,6]

Hypothermic Perfusion

(1) The low oxygen requirements of the body during differential total body hypothermia require comparatively low blood flows that could mimic the cardiac output for that particular temperature. Twenty ccs/min. per kilogram of body weight between intraesophageal temperatures of 20°C and 30°C yield adequate peripheral oxygenation, satisfactory arterial pressures, essentially unchanged electroencephalogram, and urine outputs during hypothermic total body cardiopulmonary bypass. The awakening from anesthesia is immediate and there are no abnormal neurological symptoms or signs.[3,4,6]

(2) The lower flows mean decreased trauma to blood, simpler cannulation and smaller machinery, easier to assemble and clean. The total amount of blood is reduced.

*From the Cardiovascular Section, Mercy Hospital, Oklahoma City, Oklahoma. Supported by private donations.

**Fellow in Cardiovascular Research, Mercy Hospital, Oklahoma City, Oklahoma.

Pt	Age Yrs.	Weight Kg.	Diagnosis	Flow Rate ccs/Kg. BW/min.	Total Perfusion Time min.	Duration of Aortic Occlusion min./	Temperature Range During Aortic Occlusion	Final Outcome
1	31	70	Inter-atrial defect	20	38	15*	27°C-30°C	Living and well
2	43	50	Inter-atrial defect	20	41	13*	29°C-30°C	Died 4 mos. post surgery. Autopsy: intact cardiac repair and acute necrotizing pancreatitis.
3	4	16.3	Tetralogy of Fallot	20	69	22**	26°C-30°C	Died on 2nd P.O. day after doing well. Autopsy: Partial disruption of I.V. repair.
4	14	35	Tetralogy of Fallot	20	121	66**(20, 23, 7 & 16)	27°C-30°C	Living and well
5	4	14.5	Pulmonary valvular and infundibular stenosis	20	124	0**	23.5°C-31°C***	Living and well
6	11	43	Inter-atrial defect. Expl. for pos. pulm. stenosis.	20	75	24**(18, 6)	27°C-30°C	Living and well
7	35	43	Mitral commissurotomy	20	38	18**	26°C-30°C	Living and well
8	8	19.3	Tetralogy of Fallot.	20	154	90**(15, 15, 15, 15, 15, 15)	25°C-26°C	Died on 3rd P.O. day. Autopsy: intact cardiac repair. Severe bil. pneumonia
9	9	25.56	Inter-atrial defect. Pulmonary stenosis.	20	58	12**(6, 6)	28°C-29°C	Living and well
10	5	14	Inter-vent. septal defect. (pulm. hypertensive)	20	121	40**(16,.8, 16)	24°C-25°C	Living and well
11	31	59.05	Inter-atrial defect	20	88	18**	24°C-25°C	Living and well
12	14	27.64	Inter-vent. defect. (pulm. hypertensive)	20	85	24**(18, 5)	23°C-26.5°C	Living and well
13	42	51.77	Pulm. stenosis and Infundibular stenosis	20	100	25**(5, 15, 5)	28°C-31°C	Living and well
14	7	18.96	Tetralogy of Fallot	20	116	27**(15, 7, 6)	23.5°C-24.5°C	Living and well
15	6	16.5	Tetralogy of Fallot	20	106	18**	22.25-24°C	Living and well
16	12	59.12	Pulmonary Stenosis	20	46	8*	28.5°C-29°C	Living and well
17	9	16.4	Inter-vent. septal def. pulm. hypertension.	20	80	28(3, 19, 16)*	24°C-26°C	Living and well
18	52	38.7	Mitral Stenosis and insufficiency	20	69	11*	29°C-31.2°C	Living and unchanged from pre-op. condition. Condition of the valve was such that no repair was performed
19	25	58.58	Inter-vent. septal def.	20		34** (17, 17)	25.1°C-25.2°C	Living and well

Figures between parentheses indicate single periods of aortic occlusion.
*Ventricles continued to have regular rhythm.
**Ventricular fibrillation that responded to one electroshock of 110 volts.

TABLE I. Data on hypothermic total body cardiopulmonary bypass on 19 consecutive cases.

(3) Lengthy perfusions are permitted and thorough evaluation of complicated defects accomplished.[6] Sutures are deliberately and accurately placed and patches precisely fashioned. The aim should be a lifetime cure. The continuation of perfusion for sometime after closure of the cardiac chamber may be partially responsible for the excellent behavior of the heart.

(4) Simple prolonged aortic occlusion is the easiest answer to the obtainment of an empty "motionless" heart and to prevent air embolism.[6] The electrocardiogram has shown no patterns of ischemia and the gross and microscopic detailed examination of three hearts revealed no changes compatible with necrosis, liquefaction or fibrosis.

(5) Table I summarizes 19 consecutive patients who had hypothermic cardiopulmonary bypass for open heart surgery and stresses some of the advantageous features of this technique.

Comment

Heat exchangers have been experimentally and clinically interposed in heart-lung machines for some time.[1,2,3] The simplicity, availability and efficiency are the main tributes of the one we use and has been described in detail elsewhere.[5]

The disadvantages of deadly ventricular fibrillation, limited time of venous inflow stasis, tendency to bleeding and rewarming shock of external hypothermia, and the drawbacks of high flows and increased bronchial and coronary sinus returns of normothermic perfusion are eliminated by hypothermic extracorporeal circulation. In addition, prolonged simple clamping of the aorta becomes feasible and provides the simplest way to obtain an empty "motionless" heart and to prevent air embolism. The findings of Gott et al[7] that high energy phosphates, glycogen and lactic acid of the myocardium are minimally changed during hypothermia arrested hearts corroborate our results.

ADDENDUM

Since the submission of the manuscript, this technique of open heart surgery was performed on 18 additional service and private patients without a mortality. Up to the date of proof reading (February 3, 1960), there have been 29 consecutive survivals. The completeness of the repair and back to normal life behavior are most noteworthy.

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Nazih Zuhdi, M.D.,
430 N.W. 12, Oklahoma City, Oklahoma

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The Present Status of the Internship

The internship in this country had its beginning in institutional apprenticeships, originating about 160 years ago. Official recognition first came in 1904, when the American Medical Association established standards for the internship as a part of the training of a physician. Facilities for intern training first exceeded the number of medical graduates in 1926, and stayed slightly greater than the number of graduates until just after World War II, when available positions began to increase rapidly. The number rose from about 8,000 in 1945 to 12,649 last July, at which time there were 6,860 graduating seniors from United States medical schools.

In spite of the increase in internships, the percentage of positions filled has remained at 83 per cent. Obviously only about 55 per cent of these were filled by graduates of United States medical schools. About 25 per cent were taken by graduates of foreign schools. The number of foreign graduates serving internships first became significant in 1950 and has increased rapidly since then. This rise was partly due to the passage of the United States Information and Educational Exchange Act of 1948 which facilitated the securing of visas for educational purposes. Now there is a good possibility that the number of foreign graduates available for internships will decrease because all hospitals listed for internship have agreed to accept no foreign student after July 1, 1960 who has not been certified by the Educational Council for Foreign Medical Graduates.

All of these facts show one great problem connected with the internship: a shortage of interns. Now an institution which is rooted deeply by tradition, and fixed by law, is the target for all types of recommendations for change, including abolishment.

The internship has several purposes. First it should bridge the gap between didactic instruction and practical application of the principles of medicine. Secondly, the intern learns to accomplish certain necessary procedures, to exercise judgment and make decisions, all in a setting and under circumstances where he does not have to bear the full and sole legal responsibility for his acts. Thirdly, the intern year gives the serious student of medicine a chance to decide what his future field of endeavor will be.

It must be realized that at least five parties determine the character of an internship. They are the medical student, the hospital, the Council on Medical Education and Hospitals, state boards of licensure and the medical schools. The first two parties are most directly involved and it is the balance between these two which is most important. The intern owes the hospital his loyalty and a desire to work and the hospital owes the intern educational opportunity and protection from spending too much time in tasks that have ceased to be of value in his career. This balance often is upset, with both parties being the offender.

Many solutions have been suggested for the internship problem, none of which are agreeable to everyone concerned. The least practical is abolishment of the internship, which is untenable just for the licensure problems involved.

Mark R Everett

The Normal Radiology of Old Age and Its Borderlines

ERNEST LACHMAN, M.D.

Since graduating from the Breslau Medical School in Germany in 1924, Ernest Lachman, M.D., has been certified by the American Board of Radiology. He is Professor and Chairman of the Department of Anatomy and Consultant Professor of Radiology at the Universiy of Oklahoma School of Medicine.

Doctor Lachman is a member of the American Association of Anatomists, the American College of Radiology, the American Association of Physical Anthropologists, the Society of Experimental Medicine and Biology and the Gerontological Society.

RADIOLOGY has added a new dimension to the time honored field of human anatomy by contributing a vast amount of information on normal age changes from infancy to senescence in tissues and organs that are accessible to the roentgenologic approach. For the older age groups a frame of reference can be established against which one can evaluate pathologic alterations that are the concomitant of old age. Here, as so often in radiology, the "borderlands of the normal and early pathological" (Koehler) have not been clearly defined. But most radiologists have accumulated, almost intuitively, a storehouse of visual impressions as to variations of old age, such as the increased translucency of lungs and bones, the greater number of vascular calcifications, alterations in the configuration of the spinal column and various joint changes.

While hardly any systematic presentation of these changes in the roentgenogram has been undertaken, there is nevertheless a considerable amount of information scattered through the roentgenologic and gerontologic literature. A brief review of this material seems indicated.[1]

Thoracic Cage

There is great need for vertical studies which follow healthy individuals through their life span by means of roentgenograms of the chest in one or two projections. With modern technique radiation dosage can be restricted to a minimum and the gonads protected, so that the obstacle of radiation hazard can be overcome. Until such longitudinal studies have been done, we have to be satisfied with comparative statistical appraisal of average values for different age groups. Such cross-sectional studies have been undertaken by various authors. They show that with advancing age there is a significant increase in the distance between the thoracic inlet and the dome of the diaphragm, i.e., in the roentgenographically observable height of the lungs. The maximal increase is present in the seventh decade. Concomitant with this increase and possibly causing it, flattening and lowering of the diaphragm is observed. Some investigators also demonstrate a statistically significant increase in the antero-posterior diameter of the chest or thoracic depth from the fourth decade on. Thoracic width remains stationary through-

out life. Also noted is an increase in the downward slope of the ribs, particularly in the oldest age groups. Well known is the progressive calcification of the costal cartilages with the interesting sex difference that calcification of the first costal cartilage advances much more rapidly in the male than in the female where the terminal stage of almost complete calcification is only rarely attained.

Lung and Trachea

It can be assumed that the enlargement of the thoracic cage in height and depth leads to a distention of the lung with some degree of emphysema. Indeed numerous authors have stated that emphysema is almost universal in old people. It is questionable to what extent the roentgenogram depicts minor degrees of senile emphysema. There is lack of correlation between the roentgenogram on one hand and the clinical and postmortem findings on the other. Many variables govern radiographic translucency of the lungs. These include technical factors, the depth diameter of the thorax and the degree of development of extra- and intrathoracic soft tissues. Thus increase in the over-all translucency of the lungs due to atrophy of the interalveolar septa might be counteracted by lengthening of the depth diameter of the chest, by increased filling of the blood and lymph vessels due to pulmonary congestion, and by fibrosis or bronchiectasis accompanying emphysema. The great number of variables precludes quantitative evaluation of the degree of emphysema from the roentgenogram. Other roentgenograhpic signs of emphysema, frequently found in old age, are depression and flattening of the diaphragm and restriction of respiratory excursion. There seems to be a positive correlation between advancing age and the percentage of roentgenologically detectable emphysemas so that one might almost be inclined to regard the early signs of emphysema as a physiological concomitant of senescence. Here, as in other field of geriatric roentgenology, it is difficult to draw the line between the normal involutionary process and degenerative pathology. Attention should be called to the intensification of pulmonary markings in people over 60 years of age. This intensification may be vascular or bronchial in character and may be due to pulmonary hypertension and beginning congestion or interstitial fibrosis and chronic bronchitis. A combination of all these factors is of course possible. Chronic inflammatory and cicatricial processes may lead to enlargement of the hilar shadows in advanced age. These alterations seem to be due to anthracotic and silicotic hilar sclerosis. This may occur in elderly inhabitants of industrial centers, even without occupational exposure to coal dust, since the longer life span allows for the accumulation of greater amounts of foreign material and consequent reaction to it. Frequently noted has been the gradual descent of the tracheal bifurcation with advancing age, which in old age may be as low as the seventh thoracic vertebra.

Cardiovascular System

Thoracic Aorta. There is a progressive elongation and dilatation of the aorta in the older age groups, a change which is due to loss of elasticity in the vessel wall. Consequently there is an increase in the size of the aorta as determined by roentgenographic measurements. It is also known that with advancing age more and more of the ascending aorta becomes marginal along the upper right arch of the cardiovascular silhouette in the posteroanterior view, resulting often in increased convexity of this part of the vascular contour. The aortic knob protrudes farther cranially and to the left. The left lateral contour of the descending aorta is visible to a greater length and may intersect the cardiac shadow in the region of the left auricle. Oblique views demonstrate the greater convexity of the whole thoracic aorta. Its shadow may be denser and the aortic knob frequently shows calcification.

Heart. The described changes in the aorta effect the roentgenographic appearance of the senile heart to a considerable degree, since it has to adapt itself to the available space in the mediastinum, in part preempted by the tortuous and enlarged aorta. As a consequence the heart frequently becomes more transverse. The final size, shape, and position of the "normal" senile heart is the resultant of a number of partly conflicting factors, such as the degree of

aortic dilatation, level of diaphragm, thoracic shape, presence of senile emphysema, obesity, and hypertension. Most statistical studies agree that with increasing age there is a slight increase in actual heart size, especially due to enlargement of the left ventricle. If hypertension and arteriosclerosis are marked, then the heart assumes aortic configuration which is characterized by prominence of the aortic shadow on both sides, prominence of the left ventricular contour and a deepening of the cardiac waist.

The senile atrophy of the heart with marked reduction in cardiac musculature, that is so often diagnosed by pathologists as a characteristic sign of advanced age, is generally not reflected in the roentgenogram during life because of the dynamic and variable conditions of filling. Aortic calcifications, which are a common accompaniment of old age, have been mentioned. Rarer are demonstrable calcifications in the coronary arteries and the mitral and aortic valves. The clinical value of such findings is debatable. There seems to be no correlation between the roentgenologic finding of aortic or coronary calcification and the functional effectiveness of the circulation.

Other Blood Vessels. Other vascular calcifications are also more common in the elderly. Well known is the roentgenogram of the calcified abdominal aorta, particularly on lateral views. This condition is the best clinical sign of the atherosclerotic type of arteriosclerosis and should be more frequently recognized in clinical surveys for arteriosclerosis. Rarer are calcifications in branches and sub-branches of the abdominal aorta, such as in the splenic artery. They occasionally have been mistaken for foreign bodies. More frequent are calcifications in the arteries of the extremities which appear as two main types, that of the intima and media. The intimal type is irregular with scattered discrete plaques and is often confined to certain parts of an artery and to certain sections of the vascular circumference. It is most easily demonstrated by radiographic soft tissue technique in the lower third of the femoral artery. These calcifications are a sign of arteriosclerosis and as such occur more frequently in conjunction with occlusive arterial thrombosis than with patent arteries; the roentgenographic demonstration of this type, therefore, has clinical significance.

By contrast, in calcification of the media, also known as Moenckeberg's sclerosis, the calcifications are regular, diffuse, and of fine grain and affect in general the whole circumference of the artery. There is a tendency for the calcium to accumulate in rings and to be wide-spread in character. This is the "pipe stem" type of calcification which may occur in old age without leading to obliteration of the involved vessel. It is often only an incidental finding accompanying senescence. In younger people it is an indication of disturbances of calcium metabolism.

Calcification of the internal carotid artery deserves special mention. The calcified artery, particularly if tortuous, is projected into the hypophyseal area on lateral views, which may lead to the erroneous diagnosis of calcified pituitary tumor.

The importance of calcifications of thrombi in the veins of the vesical, uterine and prostatic plexuses lies mainly in their being mistaken for ureteral or vesical calculi. The occurrence of these phleboliths is normal in older age groups.

Gastrointestinal Tract

The digestive system of the aged seems to display less wear and tear than most other systems of the human body and consequently seems capable, if not locally altered by malignancies or toxic and infectious processes, of functioning beyond the ordinary life span. Thus geriatric roentgenology of the gastrointestinal tract furnishes only a few relevant data that affect our standards for the normal adult person.

Esophagus. As far as the esophagus is concerned, the increased frequency of hiatus hernia in older age groups has often been noted, particularly in the female sex. It is particularly the sliding type of hernia without shortening of the esophagus that is apt to be found in the elderly. Loss of elasticity in the tissues around the hiatus with advancing years is apparently the cause of the condition. While there is a whole series of symp-

toms referable to hiatus hernia, it should be realized that it may be asymptomatic and only an incidental finding in a carefully performed roentgenologic examination of the gastrointestinal tract.

Stomach. So far no changes in the roentgenographic anatomy of the stomach accompanying old age have been reported in the literature. Size, shape, position and tonus of the organ seem to remain unaltered. Although there is considerable evidence that in the elderly histologic changes occur in the mucosa of the stomach, pointing in the direction of an atrophic gastritis, and that the incidence of achlorhydria increases, none of these conditions is apparently accessible to roentgenographic demonstration. The rate of gastric evacuation after a barium meal does not seem to be affected by old age.

Small Intestine. It is known that the cranial most point of the duodenum moves caudally in both sexes at a rate of half a vertebra per decade. But since the duodeno-jejunal flexure is relatively fixed, the vertical spread of the organ decreases with age. The following abnormalities have been found in a roentgen study of the small intestine in people over seventy years of age without clinical symptoms: segmentation of filling, hypertonus, hypotonus, and spasticity of portions of the intestine, hypermotility or delay in emptying. These alterations have been explained on the basis of arteriosclerotic interference with the blood supply of the intrinsic nerve plexuses of the intestine.

Colon. Roentgenologic evidence does not support the frequently expressed claim that the colonic musculature becomes atonic with advancing age and that the time of passage is delayed. But only insufficient data on the normal colon in the aged are available. Diverticulosis of the colon is a common and often incidental finding in the roentgenologic examination of older people, belonging to the borderline field of the normal.

Biliary and Urogenital Tracts

There are neither horizontal nor vertical studies available which evaluate age changes in the size, shape, and position of the gallbladder and kidney on a statistical basis. One author found considerable distension of the fundus of the senile gallbladder. On the other hand excretory studies on the gallbladder in old age seem to prove that if the biliary tract escapes pathologic alterations, its motility does not become impaired. The incidence of gallstones progressively increases with old age. They were found by one author in 70 per cent of all cases over seventy years of age not all of which of course would be demonstrable on the x-ray film. The "normal" degenerative changes, that take place in the human kidney in the process of aging, do not seem to be reflected in either a decrease in the size of the kidney shadow or in a slowing of the excretion of the contrast material in intravenous pyelography. Prostatic enlargement and diverticula of the bladder can be demonstrated radiographically by the proper cystographic procedures.

Skeleton

The skeleton of the aged displays a number of roentgenologically discernible signs of involution. Osteoporosis in older people seems to be an almost physiologic condition, caused mainly by impaired activity of the osteoblasts with resulting inadequate production of bone matrix. The disturbance manifests itself as postmenopausal osteoporosis in women past fifty and as senile osteoporosis after the age of sixty-five, the two conditions being apparently similar in origin and pathology. Anatomically, osteoporosis of old age is characterized by decrease in the thickness and number of bony trabeculae with widening of the intertrabecular spaces. The compact matter disappears more slowly. In milder cases osteoporosis is limited to spinal column and ribs. Further advance results in involvement of pelvis and proximal femura, leading finally to inclusion of the whole skeleton. It requires the disappearance of fairly large amounts of calcium, before the diagnosis can be made roentgenographically. The minimum amount of decalcification necessary for roentgenological visibility has been variously given as 20-60%. It has been estimated that more than half of all women over the age of fifty and more than half of both sexes past sixty show a sufficient degree of osteoporosis to be roentgenologically demonstrable.

Early roentgenographic signs of senile osteoporosis may not be easily recognized; in fact minor decrease in bone density may be ascribed to technical variations such as overexposure or over-penetration. In more marked cases the trabeculae are clearly scarcer and thinner than normal, with widening of the trabecular meshwork. Later the cortex also becomes thin and lamellar with increase in the width of the medullary cavity, and it apears as if drawn with a fine pencil. Finally, trabecular structure may become almost invisible and the bone takes on a glass-like appearance.

Senile osteoporosis is most easily demonstrated roentgenographically in the spinal column. The bone texture appears as previously described, with the added feature of asymptomatic collapse of one or more vertebrae. The "codfish" appearance of the spinal column with over-all decrease in density, with ballooning of intervertebral disks, and biconcave deformity of the vertebral bodies offers a picture that is often in startling contrast to the scarcity of symptoms. Clinically undiagnosed fractures occur in as many as 25% of all cases. The lower thoracic and upper lumbar segments of the column are most commonly affected by these changes, while wedging of the vertebrae as a result of osteoporosis involves mainly the mid-thoracic vertebrae and may lead to marked kyphosis of this region. This osteoporotic type of senile kyphosis has to be differentiated from ordinary kyphosis of old age. While the site of both deformities is the same—the mid-thoracic region—their anatomy is quite different. In the osteoporotic form the kyphosis is the result of a wedge-shaped collapse of one or more vertebrae, while in the ordinary type of deformity is brought about by degeneration and partial disintegration of the disk with preservation of the quadrangular form of the vertebral body. For static reasons mainly the anterior portions of the intervertebral cartilage are involved, which in the final stage are completely replaced by a bony bridge between adjacent vertebrae. In the oldest age groups there is often a combination of the two types.

In the pelvis and upper femur the disappearance of part of the spongy matter may result in coarsening of the functionally most important trabeculae which—due to thinning of the cortex—stand out quite distinctly. Structural weakening of the bone often leads to decrease in the angle between the axes of the neck and shaft of the femur with advancing age. This, and the changes in the spinal column, are the reasons for decrease in height in older individuals.

The other long bones as well as the skeleton of hand and feet may display similar changes consisting of cortical thinning, widening of the trabecular meshwork and over-all decrease in bone density. The roentgen signs of senile osteoporosis may remain stationary for many years and may be unaccompanied by any clinical symptoms, as stated before.

A second group of bony changes, associated with advancing age and on the borderline of the normal, are alterations along the articular surfaces of long bones, vertebral column and ribs. They are the result of degenerative changes in the cartilage which, on the basis of chronic wear and tear and decreased blood supply, loses its elasticity. The bony changes comprise reactive ligamentous ossifications and new bone formation at the capsular attachment of the joint. Anatomically and roentgenographically the process assumes the form of marginal osteophytes, spurs and lipping; clinically it is described as senescent, degenerative, hypertrophic or osteoarthritis. The condition seems to be universal after the age of forty-five and progresses with advancing years. The heavier constitutional type is apt to acquire these changes earlier than the slender type.

There are hardly any roentgenologic studies available on the distribution and frequency of this condition throughout the skeleton. But we know that the articular surfaces of the vertebral column, of pelvis and knee joint are most apt to reveal roentgenographic changes, although the joints of the upper extremity, likewise, do not escape. In the knee joint, in addition to narrowing of the joint space, spur formation and lipping, we find occasional asymptomatic calcification of the menisci. Spurring on the calcaneus also is stated to be more common

in older age groups. Quite interesting is the observation that the frequency of sesamoid and accessory bones increases with advancing age.

Head and Neck

Numerous are the changes of old age in the roentgenogram of the skull. Senile atrophy is quite common, leading frequently to a "washed-out" appearance of the calvarium, which may be uniform or spotty and which has to be differentiated from ostitis fibrosa cystica and the early stages of Paget's disease. In one series two-thirds of the aged subjects showed roentgenographic evidence of minimal to moderate osteoporosis of the skull. The sella turcica is one of the first regions to reveal these changes. The dorsum sellae appears thin and translucent with narrowing also from side to side, alterations that have to be distinguished from localized pressure erosions. The over-all glass-like translucency of the sellar area contrasts sharply with the frequently present calcifications of the superimposed internal carotid artery. As far as the paranasal sinuses are concerned, some diminish in size in old age, particularly the maxillary sinus, while the frontal and sphenoidal sinuses increase in extent.

In regard to the roentgen pattern of the diploic veins, the literature contains conflicting statements. While one group of authors speaks of regression with age, others mention enlargement and widening of the diploic canals. The depressions caused by arachnoid granulations become progressively larger with advancing age although there are marked variations from individual to individual.

In contrast to the common appearance of osteoporosis of the senile skull, a hyperostosis of the cranial bones is rather rare. This is located in the anterior cranial fossa, involving the frontal bone, and may be diffuse or circumscribed and nodular. It represents a condition on the borderline of the pathologic and occurs mostly in women past middle age.

The cranial sutures are as variable as other skull markings mentioned so far, although there is a typical picture represented by complete closure of all sutures in old age. In general the coronal suture is almost invariably closed in older age groups, the sagittal suture in women past fifty and in men past sixty; the lambdoid suture may remain open throughout life in a fair sized number of cases and this may occasionally happen also in other sutures. Where sutures are obliterated, their sites may be indicated by a line of denser sclerotic bone.

Omitting dental and alveolar changes from this discussion, one normal finding in the skull of the aged remains to be mentioned, i.e., the calcifications in the pineal body, in the falx cerebri, in the margins of the diaphragm of the sella turcica, in the free edges of the tentorium cerebelli and posterior to the dorsum of the sella and in the choroid plexus, in addition to the calcifications in the internal carotid artery mentioned previously. While calcifications in the pineal body may occur in younger years and even in adolescence, they reach a frequency of 78% in people past sixty. They have been found in 55% of all individuals past twenty-five.

Age changes in the form of laryngeal calcifications were known even before the roentgen era. While ossification of laryngeal cartilages starts around the age of twenty, it progressively increases, reaching its maximum extent around the sixty-fifth year. Calcification may also involve the tracheal rings. In the larynx, as so often, there are many variations in the aging process even to the extent of complete absence of calcifications in the older individual. One author found that there is a definite relation between the degree of ossification of laryngeal cartilages and the presence of mineral matter in the wall of the aorta.

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FOOTNOTE

1. For a detailed bibliography see the author's paper listed under "References."

801 N.E. 13th, Oklahoma City, Oklahoma

Etiology of Peptic Ulcer Disease

Editor

THOMAS N. LYNN, M.D.
Chief Medical Resident, University Hospital, Oklahoma City, Oklahoma

Discussants

JERRY BRESSIE, M.D.
Medical Resident, University of Oklahoma Medical Center

WYMAN EWING, M.D.
Radiology Resident, University of Oklahoma Medical Center

JACK WELSH, M.D.
Instructor in Medicine, University of Oklahoma Medical Center

LEE HONSKA, M.D.
Instructor in Medicine and Preventive Medicine, University of Oklahoma Medical Center

Doctor Jerry Bressie: The patient for this morning is Mr. M. P., a 20 year old white male who was admitted on 11-10-59, complaining of vomiting coffee ground material and having tarry stools on the day of admission.

PRESENT ILLNESS: This 20 year old white male filling station employee first became ill at the age of 12 years, having sharp, severe periumbilical pain which disappeared after about one week, but which reappeared, and has continued to be intermittantly present since the age of 16 years. These were not typical "ulcer pains" but nonetheless the patient had a perforation of an anterior duodenal ulcer at the age of 18 years, which was repaired at University Hospital. He was then placed on management consisting of frequent feedings and antacids which he stated that he followed religiously. Nevertheless in April, 1959, he had the acute onset of hematemesis which required the replacement of four units of blood. He was treated conservatively and remained on an ulcer program but continued to have the periumbilical cramping every two or three weeks. He did, however, note transient relief of this with the intake of alkali and antispasmodics. Approximately 4:00 p.m. on the day of admission he had the onset of nausea and promptly vomited approximately one pint of coffee ground material, shortly followed by the passage of black feces. On his arrival at University Hospital he vomited blood a second time. The past history and review of systems is non-contributory.

SOCIAL HISTORY: The social history is of interest in that the patient quit school in the 10th grade. He has been employed at many jobs, usually as a farm laborer and more recently as a filling station attendant. It is noteworthy that he has had a bleeding episode or exacerbation of the ulcer pain following each new employment. He feels that he is a failure in life and now aspires to employment of a more technical nature, which would require a period of special training which he cannot undertake due to the responsibilities of a home and family. He feels that he is now destined to remain in a more or less menial job for the remainder of his life.

PHYSICAL EXAMINATION: The blood pressure on admission was 110/70; pulse 120 per minute; respiration 16 per minute.

He was quite pale and appeared acutely ill. The significant physical findings were otherwise limited to the abdomen, where no guarding or rebound tenderness was elicited. Bowel sounds were present. The liver was down one cm. beneath the right costal margin but no other viscera or masses were palpable.

LABORATORY STUDIES: Laboratory studies on admission revealed a hemoglobin of 9.5 gms.% with a hematocrit of 32%. The white blood count was 15,700 per cubic millimeter with a differential of 47% polymorphonuclear leukocytes, 43% lymphocytes, 4% monocytes, 3% eosinophils and 2% basophils. Bleeding and clotting studies were normal as was the urinalysis. A stool examination on admission was positive for blood, however by the sixth hospital day the stools had become negative. The alkaline phosphatase was 1.7 Bessie Lowery unit, transaminase studies were within normal limits, BUN was initially 33 mgs.% but by the sixth hospital day, had fallen to 14 mgs.%. The fasting blood sugar was 100 mgs%. Calcium and phosphorus determinations were within normal limits. The gastric analysis showed a total volume of 175 cc. with a free acid of 70 degrees and a total acid of 80 degrees.

HOSPITAL COURSE: The patient was treated initially with whole blood transfusions and following this showed no further evidence of significant bleeding. He has subsequently been given frequent feedings and interval antiacids. He is at present asymptomatic and is eager to leave the hospital.

Doctor Wyman Ewing: A single PA view of the chest shows a normal heart and lungs and the visualized osseous structures appear normal. We also have an upper GI series which surprisingly enough shows a normal esophagus, stomach, duodenum and proximal small bowel. These films are perfectly normal.

Jack D. Welsh, M.D.: Any attempts to present an orderly review of the theories regarding the etiology of peptic ulceration are destined to frustration. This is in part due to the great quantity of recorded experimental data and philosophical ideas and to the conflicting interpretations of these data. In addition, difficulty is encountered in the clear separation of "primary causes" from "contributing factors." With these limitations in mind, I shall attempt to outline the major theories of causation that have, at one time or another, been popular in the medical world.

The conditions considered a possible cause of, or contributing factors in the genesis of peptic ulcers may be separated into 12 general categories. I would like to review these briefly before we consider present-day theories. No attempt will be made to present all of the available data concerning each of these, or to mention all of the individuals concerned.

Review of Theories

1. *Vascular:* Rokitansky, as early as 1849, thought the etiology of peptic ulceration of the upper intestinal tract was due to hemorrhagic erosion. The idea of vascular embolism, thrombosis, and infarction causing the ulceration was proposed by Virchow in 1853. Peculiarities of the blood supply, atherosclerotic changes, and more recently, arterial spasm, and alteration in shunt systems have also been implicated.

2. *Mechanical:* Aschoff suggested that mechanical factors were of importance in producing ulcers. He proposed that such ulcers occurred along the lesser curvature in the so-called Magenstrasse, in areas which are most exposed to traumatization from the acid and gastric contents. Other mechanical factors that have been proposed are: chronic compression of the abdomen, blows to the abdomen, rough foods, spasm and hyperperistalsis.

3. *Infection:* Various observers have considered foci of infection to be important. This thesis was particularly championed by Rosenow from 1913 to 1923. Recently, less credence has been given to the focal infection theory; however, the frequent association of ulcers and respiratory infections is well recognized.

4. *Chemical:* Cohnheim, as early as 1880, and Regal in 1886, proposed the idea of hydrochloric acid causing ulcers. In the early 1900's, many workers such as Hurst,

Stewart, and Sippy, stressed the role of hydrochloric acid and pepsin. Since these factors are still considered to be important, they will be discussed later in more detail.

5. *Neurogenic:* The relationship of intracranial lesions and acute ulceration of the upper intestinal tract has been noted by such men as Rokitansky, Voderahe, Cushing, and others. These cases, however, form a special group and probably have little or no relationship to the type of ulcer being discussed today. Extensive data have accumulated on the influence exerted on gastric secretion and motor function by the accelerator influence of the vagus and the inhibiting influence of the sympathetic nervous system. Von Bergmann felt that a functional disharmony might exist between the vagus and sympathetic nerves thereby causing spasm of the musculature and vessels of the stomach and duodenum, this in turn leading to submucosal ischemia, erosion, and finally to ulceration.

6. *Allergy:* The ability to produce allergic lesions in the intestinal tract of animals and the reduction of symptoms in some patients by elimination diets, have caused some to believe that peptic ulcers have an allergic origin. Although this may be a contributing factor in rare cases, its importance in the genesis of the majority of ulcers is doubtful.

7. *Constitutional and predisposing factors:* Much has been written concerning the "type" of individual who develops an ulcer. This will be discussed later.

8. *Gastritis:* The role which gastritis plays in the pathogenesis of ulcer remains highly controversial. Erosion and acute ulcers of the stomach are common accompaniments of chronic gastritis. Whether this pertains in duodenal ulcer has not been established. Conversely, however, there is ample proof that intermittent pylorospasm and pyloric obstruction may bring about secondary gastric changes.

9. *Nutritional deficiency:* Diets high or low in protein, high or low in carbohydrate, or diets deficient in fat or vitamins have been blamed for the production of peptic ulcers. The only definite conclusion is that animals and man can develop ulcers on abnormal diets.

10. *Endocrine:* The possibility of endocrine imbalance leading to ulceration of the gastrointestinal tract is not a new consideration. It has received new impetus over the last few years with the demonstration of ulcerations associated with tumors of the pancreas and other endocrine glands. Recent studies, beginning with Selye's introduction of the "general adaptation syndrome," would suggest that the adrenal glands may participate in the pathogenesis of peptic ulcer.

11. *Intragastric mucosal irritants:* Experiments with corrosive substances in animals, the high incidence of peptic ulceration in Abyassinia and India where the natives' diet is generously seasoned with pepper, and many other such observations have led some to the conclusion that irritants in the food are an important factor in the genesis of ulcers. It is questionable, however, whether the experiments in animals are comparable with the conditions in humans. Also, many of the groups whose diets contain large amounts of condiments have associated nutritional deficiencies.

12. *Occupational:* I have mentioned this only for completeness since Doctor Honska will discuss the influence of occupation in more detail.

Modern Concepts

Even with our present "sophisticated" approach to peptic ulcer disease, the literature is filled largely with information obtained through a gastric tube or from an x-ray machine. Since the x-ray evidence of ulceration of the duodenum of the patient presented today was debatable, I think it is relevant to quote from an article, "The Trouble We're in Over Duodenal Ulcer," by Eddie Palmer.[1] "Upsetting as it may be, it is necessary to admit that the reason is that, of the complex total-patient disease which 'ulcer' is, only the little crater can be measured by an objective device, a device which doesn't require much thinking—or at least much assumption of critical diagnostic responsibility—on anybody's part. Professional insecurity being what it is, it sometimes seems best to wait for the x-ray machine to tell what the prob-

lem is or even whether the patient is sick or not. This misses the point of ulcerative disease, because it really doesn't matter very much whether the little crater is there or not. Among the things that the patient with ulcerative disease is likely to show are narrow subcostal angle, a high arched palate, and a crater in the duodenum—but not always. And, because anatomy permits it, the crater comes and goes—not as the indication of a disease, but in response to the disease. The patient isn't sick because he has an ulcer, but has an ulcer because he is sick."

Most workers in the field today question whether just one factor is responsible for all peptic ulceration. Rather, it is felt that an interplay of several factors, varying in different persons and even in the same individual under changing circumstances contributes to the production of peptic ulcer disease. These factors can be divided into two main groups, each with their own subdivisions. (1) Local factors. (a) Eroding factors. (b) Local tissue resistance, (defense mechanisms, and ability to heal). (2) General factors. (a) Physiologic. (b) Genetic or constitutional proclivity. (c) Psychologic. (d) Ecologic or environmental. Present evidence would suggest that these may be interrelated, at least to some extent, by neural, and hormonal factors.

(1) *Local*

a. *Eroding factors:* It is well known that if there is no acid there is no ulcer.[2,3] Some feel that the acid is probably more important than the pepsin in the production of the lesion,[3] however, both are necessary. Considering duodenal ulcer patients as a group, they are hypersecretors, have increased gastric acid and pepsin, respond more than non-ulcer patients to secretory stimulating agents, and their night secretion is greater than in normals or patients with gastric ulcers.[3-7] Taken individually, however, some duodenal ulcer patients secrete less than normal persons,[3] even when the ulcer is in an active phase.[8] In the duodenal ulcer patient who has hypersecretion, it is continuous and the stomach doesn't get a "breathing spell" or rest from this high acid-pepsin bath. Experimentally it has been shown that the continued dripping of excess acid into the stomach of dogs depresses the neutralizing ability of their stomachs.[9] We do not know whether this happens in man; however, it is possible that in some patients the continued hypersecretion of acid may do the same. A look at the other side of the coin reveals that the magnitude of the hypersecretion is not enough to be the sole cause of the ulcer.[3,10] The secretory activity of patients with duodenal ulcer is the same whether or not there is an active ulcer. In other words, the ulcer can come and go while the stomach is at the same high secretory level.[3,8,11,12] Therefore, if the secretory level were the sole factor in producing the ulcer, it should wax and wane as does the x-ray evidence of the ulcer crater.

b. *Local tissue resistance, defense mechanisms, and ability to heal:* Gastric biopsy of another area of the stomach at a time when an active ulcer is present will heal in about two days without producing an ulcer. What prevents the healing of one break in the mucosa and not the other is an interesting question on which to speculate. This has been thought to be anatomical and related to the trauma caused by the passage of food or to the jet of gastric juice passing from the stomach and striking a localized area in the duodenum. There has also been shown to be a gradient of susceptibility to the eroding effects of an acid-pepsin mixture in the digestive tract.[10] Vascular changes may influence this local tissue resistance and, as we mentioned, earlier workers felt that this was related to endarteritis and other changes leading to ischemia. However, Wolf and others have shown that hyperemia precedes the production of petechiae, increased fragility, and ulceration.[13] Recent work with microradiographs has increased our knowledge of the shunting systems in the mucosa and submucosa in the intestine. It has been shown that vagal stimulation closes these shunts and can increase blood flow to the mucosa thereby producing plethora and possibly decreased tissue resistance. If this is a factor, why it occurs only in such a localized area has not been explained, but apparently vascular changes may be important.

(2) *General*

Since any division of these factors is ar-

tificial because of their inter-relation, it will be necessary to refer back to the local conditions in the discussion of general factors.

a. *Physiologic:* The upper intestinal tract appears to have a proclivity to respond to different noxious stimuli by ulcerating.[14] This has been well documented both experimentally and clinically. First, acute ulceration of the upper intestinal tract can be produced quite readily with a number of noxious stimuli such as histamine in bees wax, adrenalin, pitressin, and so forth. Second, individuals who have never had ulcers frequently develop them when they are subjected to the stress of infections, intracranial lesions, burns, or severe trauma. Why this occurs is unknown; however, the work of Selye and others would suggest that it is related to the "general adaptation syndrome."[14]

b. *Genetic proclivity or the constitutional factors:* The early work which attempted to relate peptic ulceration to certain body-types is interesting, however it is of little help in understanding the genesis of the disease. Recently, more emphasis has been placed on the frequent strong family history in these patients, and the increased incidence of ulceration in patients with type O blood. It has also been demonstrated that patients with duodenal ulcers have a greater secretory cell mass of their stomachs than healthy subjects,[7] and that there is a positive correlation between the number of parietal cells of the stomach and the presence of duodenal ulcer.[15] In summing up some of the more current ideas on the constitutional or possible genetic factors, it can be said that duodenal ulcers tend to occur more frequently in individuals with type O blood and a family history of ulceration, these patients have an increased secretory mass and number of parietal cells of their stomachs, and in some, the expected depression of gastric secretion following a duodenal meal is absent.

c. *Psychologic:* Doctor Wolf and others have shown that meaningful situations which are stressful to the individual will cause gastric hyperfunction, decreased pain threshold of the stomach, and hyperemia with increased fragility of the mucosa.[13] Two studies by Mirsky help illustrate the interrelationship of the physiologic, psychologic, and social factors.[16] He feels that the gastric hypersecretion may be a predisposing factor and not an immediate causative factor. He used the serum pepsinogen level as an index of gastric secretion and in the first study he obtained pepsinogen levels on 2,073 draftees. Subjects with values greater than one standard deviation of the mean value of the adult healthy population were considered hypersecretors. Those with values below one standard deviation of the mean were regarded as hyposecretors. In this study, the 16 weeks of army basic training was used as an environmental situation which might prove stressful to some and not to others. He obtained complete gastrointestinal x-rays and psychologic tests on 63 of the hypersecretor group and 57 of the hyposecretor group. This first roentgenologic examination revealed evidence of a healed duodenal ulcer in three men and an active ulcer in one, all from the hypersecretor group. The examinations were repeated on all but thirteen men between the eighth and sixteenth week of training. Evidence of an active duodenal ulcer was found in an additional five individuals whose original x-ray studies were negative. They were also from the hypersecretory group. Therefore, nine of the individuals in the hypersecretory group were found to have or to have developed an ulcer, whereas none of the patients in the hyposecretory group did. In the second study, which is a long-term study and as yet incomplete, serum pepsinogen levels were determined on 1,600 children, 16 years of age or less, and 4,460 adult men and women. The 2% with the highest levels were considered hypersecretors and the 2% with the lowest were considered hyposecretors. During the several years that this study has been conducted, a fairly large number of "healthy" hypersecretors have developed duodenal ulcers while none of the hyposecretors have. In this group, the noxious stimulus occurred spontaneously in their everyday life and, in most cases, it was possible to determine this significant event.

d. *Ecologic:* This is the subject of the talk by Doctor Honska and I shall omit it from my discussion.

Even from this oversimplified and abreviated presentation on the possible etiologies

of peptic ulcer disease, it is apparent that in each case, there are probably many causative and contributing factors. This is important both to the research worker who might measure only one or two parameters and the clinician who has a fixed therapeutic regimen for each patient. We might learn more about this disease and our patients if we ignored the tendency to catalogue all the historical, laboratory, and therapy response data, and studied each patient as a person with certain individual psychologic, physiologic and therapeutic responses. A classification of this type would be more meaningful than the presence or absence of a "little crater."

Lee Honska, M.D.: The duodenal ulcer patient may classically be described as a long, thin, hungry type with intense anxiety, insecurity, resentment, guilt and frustration. The problem of peptic ulcer is wide spread throughout the world. Of interest is the fact that the pattern of the disease varies from place to place and from time to time in incidence and various characteristics. In North England duodenal ulcer is more common than in South England. There is more seasonal variation in ulcer disease in the United States than Brazil. In the 19th Century duodenal ulcers were more common in women and now they are common in men.[17] In discussing the epidemiology of peptic ulcer I will try to point out some of these problems.

Peptic ulcer occurs more frequently in urban than in rural areas. Davies stated that ulcer is unknown in primitive uncivilized races, however, I might add that he forgot to mention that gastroenterologists and x-ray machines are also unknown in these areas. Autopsy figures suggest that 10% of all people suffer from a duodenal or gastric ulcer sometime in their life. Although an ulcer may occur at anytime the highest incidence occurs between 17 and 50 years and Caucasians are affected more than the colored races.

In regard to sex, duodenal ulcer with its complications of perforation and hemorrhage occurs more frequently in males than in females, a ratio of 10 to 1.[18] This is an interesting change since the beginning of the 19th Century when peptic ulcer was more common in women.[19] This change has occurred in Germany, France and all English speaking countries.[20] During the 19th Century a woman's opportunities were limited. Success usually meant getting married and this had to occur between 19 and 30 years or she was a failure. She had to remain sweet and mild and fainting was the only outlet for her emotions and hostilities. With the 20th Century, a women's place in life has changed. If she works and is a failure she always can retreat back to the home without disgrace. Men, on the other hand, have had no change in their responsibility and frequently have lost the emotional support from the wife at home as well as having increasing competition with her at the office. Thus, it may be that there is not a true increase in the number of peptic ulcers in men but a relative decrease in those of women.

Genetic factors probably play some role in peptic ulcer disease. There are numerous examples of several cases occurring in the same family and to support the genetic theories is the fact that the people with group O blood are 35% more likely to have peptic ulcer than patients with other blood groups.[21] Doctor Welsh has mentioned other evidence supporting the hypothesis that there is a genetic aspect of the disease.

Environment has been discussed in relation to peptic ulcers. In Brazil where the temperature is more constant during the year, we do not see the seasonal variations reported in this country.[17]

Boles and Westerman[22] have correlated temperature changes with hemorrhage from duodenal ulcer and showed that this occurs more often when the temperature changes are the greatest; November, December, March and April. We also know that more people perforate their ulcer on Friday and Saturday and after 4 p.m.

In regard to habits, smoking has been incriminated, but the only thing that is known is that more ulcer patients smoke, and smoke more than the general population and that this makes very little difference in the healing rate of the ulcer.[23]

The socio-economic factors of peptic ulcer are interesting and evidence may be produced to prove any question. Sarah Jordan reports a higher incidence in the higher economic class and Doctor Jones reports a higher incidence in the lower economic class. It would appear that this difference depends on the type of patient they see.

Occupation has been extensively studied in patients with peptic ulcer. Porter[24] has reported some interesting findings in his work with monkeys. One monkey (his executive monkey) was given the responsibility of pushing a bar to prevent a control monkey and himself from receiving a shock. The executive monkey developed an ulcer, the control monkey did not. His conclusion from this was that it is "not the amount of work that is involved but the amount of responsibility," or transposing this to humans, the amount of responsibility the patient feels.

Certain other diseases have been related to peptic ulcer. Emphysema, cirrhosis and coronary disease[25] each show a relatively high correlation with the presence of duodenal ulcer. But similarly almost every other disease has also been associated in some degree with peptic ulcer by someone.

At the recent World Congress of Gastroenterology, experts from all over the world discussed the epidemiology of peptic ulcer. With understandable patriotism, the Indian absolved spices; the Frenchman, wine; the Brazilian, coffee. Others made positive correlations and stated that ulcers reappeared with the seasons, with the resumption of smoking, with overeating, with undereating, with making too much money, with having too little money, and naturally with emotional stress.

CLOSING REMARKS by Stewart Wolf, M.D.

We are very much indebted to Doctors Welsh and Honska for their scholarly exposition of the many aspects of peptic ulcer disease. They have made it clear that there is still a great deal to learn about gastric secretion and the vulnerability of the duodenal mucosa. They have both implied to us, and I think their implication is attractive, that in searching for the cause of a disease we search for the will-o'-the-wisp. It would appear that there are many circumstances which enter the picture and ultimately determine sickness or health. Among these are the inherent characteristics of the person, his way of life, and the multitude of hazards that he may encounter along the road. As soon as we give up the idea of a single cause and concern ourselves with relevant factors, our understanding of peptic ulcer, and indeed of other disease processes, will be enhanced.

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Thomas N. Lynn, M.D.
800 N.E. 13th, Oklahoma City, Oklahoma

ABSTRACTS

X-Ray Studies in Establishing Identity and Manner of Death

ERNEST LACHMAN, M.D.*

The Journal of Criminal Law, Criminology and Police Science, Vol. 50, No. 2, July-August, 1959

X-rays are an indispensable tool of the crime detection laboratory. The roentgenographic method permits us to verify or exclude the human origin of parts submitted and to determine the approximate age of a cadaver, particularly under 25, without destruction of its soft tissues. Roentgenographic study of the fetal skeleton allows the determination of a viable age and of full term, roentgenograms of the adolescent hand can predict menarche within a range of six months. After 25 years of age, closing of cranial sutures, presence or absence of teeth, progressive calcifications in the costal cartilages and secondary changes in the joints serve as less reliable age indicators.

Identification of the individual x-rays is based on skeletal features typical for the individual as established by previous roentgenograms, such as size and shape of the paranasal sinuses, congenital deformities, old fractures, curvatures of the spine, residues of previous illnesses and minor features of individual bones.

As to determination of manner of death, the demonstration of vital fractures, and presence and direction of bullets including their point of entrance are within the province of roentgenography. Numerous examples from the author's experience and the literature illustrate the discussion.

*Professor and Chairman of the Department of Anatomy.

Relative Potencies of Chlorpropamide And Tolbutamide in Man

KELLY M. WEST* and STANLEY R. McCAMPBELL**
Annals of The New York Academy of Sciences **74:** 473-477, March 1959

Chlorpropamide, 1 gm., produced significantly greater hypoglycemic responses in normal subject one hour after ingestion than did tolbutamide, 1 gm. Chlorpropamide, 0.25 gm. and 0.5 gm., produced hypoglycemic responses in normal subjects two hours after ingestion comparable to doses of tolbutamide twice as great. Since is it unlikely that either drug had been appreciably metabolized or excreted one to two hours after ingestion, the relatively greater potency of chlorpropamide probably is not attributable entirely to its longer biological half time.

Seven normal subjects had serum chlorpropamide levels averaging 11.5 mg. per cent two hours after ingesting chlorpropamide, 1 gm., indicating that about one-third of this dose was present in the plasma at that time.

Chlorpropamide, 0.5 gm., significantly increased the rate of disappearance of intravenously injected glucose. In one subject the effect of an intravenous injection of chlorpropamide, 0.5 mg., on the glucose disappearance rate was strikingly similar to that of 1.8 units of insulin administered intravenously.

Two findings that have resulted from studies carried out since this report was drafted are of considerable interest in connection with the initial findings. We found that serum levels of chlorpropamide two hours after the drug was administered orally were much higher than serum levels of tolbutamide after comparable doses of that drug. We found also that when chlorpropamide and tolbutamide were administered intravenously the hypoglycemic effects during the first hour were not significantly different.

*Assistant Professor of Medicine.
**Instructor in Medicine and in Preventive Medicine and Public Health.

The Comparative Pharmacology of Tolbutamide, Carbutamide, Chlorpropamide And Metahexamide in Man

KELLY M. WEST* and PHILLIP C. JOHNSON**
Metabolism, **8:** 596-605, July 1959

The absorption of orally administered chlorpropamide and metahexamide in man is rapid and is usually virtually complete in two to three hours. The absorption of carbutamide is somewhat slower, and the absorption of tolbutamide occurs much more slowly. Absorption of each of these four drugs is eventually virtually complete.

We found no evidence of differences in the potencies of tolbutamide, metahexamide and chlorpropamide in acute experiments in which serum levels of comparable magnitude were produced after oral administration. However, hypoglycemic responses of normal subjects to metahexamide immediately after intravenous administration suggested that it was about three to four times as potent as tolbutamide.

The serum levels of tolbutamide, metahexamide, carbutamide and chlorpropamide after intravenous injection are much too high to be compatible with the concept that they are uniformly distributed in the extracellular fluids. Direct measurements of tolbutamide, chlorpropamide and carbutamide in extracellular fluids other than serum such as edema, ascitic and pleural fluid indicate that levels in these extracellular fluids are considerably lower than in serum.

The serum levels of tolbutamide, chlorpropamide and carbutamide 40 minutes after an intravenous injection

are virtually identical, suggesting that there are no substantial differences among these three compounds in respect to the volume in which they are distributed in the body.

*Assistant Professor of Medicine.
**Associate Professor of Medicine.

The Intravenous Glucose Tolerance Test

KELLY M. WEST* and DOROTHY ANTONIA WOOD**

The American Journal of the Medical Sciences 238: 25-37, July 1959

When a glucose load of a standard size is administered rapidly intravenously, the rate of fall of the blood glucose during the first 25 minutes after the injection may be influenced by several factors which are not related to the rate of glucose utilization. Some of these factors are the size of the glucose space (which is in turn related to body size), the height of the blood glucose before the injection, the renal excretion for glucose, and the size of the glucose load.

In normal subjects, the rate of disappearance of the blood glucose after a rapidly administered glucose load of 25 gm. usually remains exponential in regard to time during the period 25 to 60 minutes after the injection at a time when the blood glucose is falling from levels near the renal threshold for glucose to levels at or just above preinjection levels. It seems likely that during this period the rate of disappearance of the absolute glucose is well related to glucose utilization although some other factors are probably operative. On the other hand during this same period the rate of disappearance of the increment blood glucose (or glucose excess) is inconstant in that the log plots of these values against time form a convex rather than a straight line. Evidence is presented which suggests that the advantages of analyzing increment rather than absolute values which have been described previously were apparent rather than real.

Glucose tolerance can be measured by administering a load rapidly intravenously of sufficient size to produce the hyperglycemia of a "physiologic" magnitude and by determining the rate of fall from a time the blood glucose is near the renal threshold for glucose until it is approaching the preinjection level. Evidence is presented suggesting that under certain conditions, fairly accurate estimation of glucose tolerance may require only one or two determinations of blood glucose.

*Assistant Professor of Medicine.
**Medical Technologist, Experimental Therapeutic Unit.

The Tuberculin Test

HARRIS D. RILEY, Jr.*

American Practitioner and Digest of Treatment 10: 1487-1488, September 1959

*Professor of Pediatrics.

Expeditious Evaluation of Circumscribed Pulmonary Shadows

ALLEN E. GREER,* JOHN M. CAREY** and NAZIH ZUHDI***

The Journal of the AMA 171: 1783-1786, November 1959

Of 85 patients with asymptomatic pulmonary rounded opacities one-half had neoplasms and over one-third had primary carcinomas of the lung. Since the most important consideration is whether these lesions are benign or malignant, early exploration is recommended.

*Assistant Professor of Surgery.
**Instructor in Surgery.
***Resident in Thoracic Surgery.

Treatment of Diabetes Mellitus With Orally Administered Compounds

KELLY M. WEST* and STANLEY R. McCAMPBELL**

Southern Medical Journal, Journal of the Southern Medical Association 52: 655-658, June 1959

*Assistant Professor of Medicine.
**Instructor in Medicine and in Preventive Medicine and Public Health.

Appointments Announced By Board of Regents

John W. Kelly, Ph.D., former member of the Medical College of Virginia, has been appointed an associate professor of pathology at the University of Oklahoma School of Medicine.

Teaching in the anatomy department of the Virginia college from 1951 to 1958, Doctor Kelly spent one year as a guest investigator at the Institute for Cell Research, Medical Nobel Institute, Stockholm, Sweden. He was a John Simon Guggenheim fellow.

Doctor Kelly received his Ph.D. from the University of Pennsylvania. He is studying the histochemistry of mucopolysaccharides and of anionic disease dyes.

University regents also have approved appointment of the following to teach on a part-time basis: Harry Martin, Ph.D., staff clinical psychologist at the Oklahoma City Veterans Hospital, instructor in psychiatry; Galen P. Robbins, M.D., former assistant in medicine and fellow in cardiology at Baylor University College of Medicine, Houston, clinical assistant in medicine; Walter Robert Cooper Jr., D.D.S., periodontist formerly of Tulsa, clinical assistant in oral surgery.

PRESIDENT'S LETTER



No More Lincolns

Would one of history's most revered citizens have risen from a humble beginning to a position of adulated stature if he had lived in today's welfare environment?

With this challenging thought in mind, Abraham Lincoln's birthday takes on more significance than its usual role of routine public acceptance as just another national holiday, or, in many cases, simply another day of relief from productivity.

It seems fair to assume that a mid-twentieth century Abe would have been deterred, if not completely thwarted, in his efforts to shake off the dust of illiteracy, poverty and obscurity. The offspring of an impoverished itinerant farmer has little opportunity today to even recognize the values of personal initiative and enterprise, much less have the old fashioned gumption to strive for such qualities.

A combination of welfare checks, subsidies and land banks would cast a paternalistic shadow of inertia over such unprofitable and ignominious pastimes as splitting rails. And would Abe have read his books by firelight, or would he have sat idle and complained that the REA was denying him his birthright of electric power?

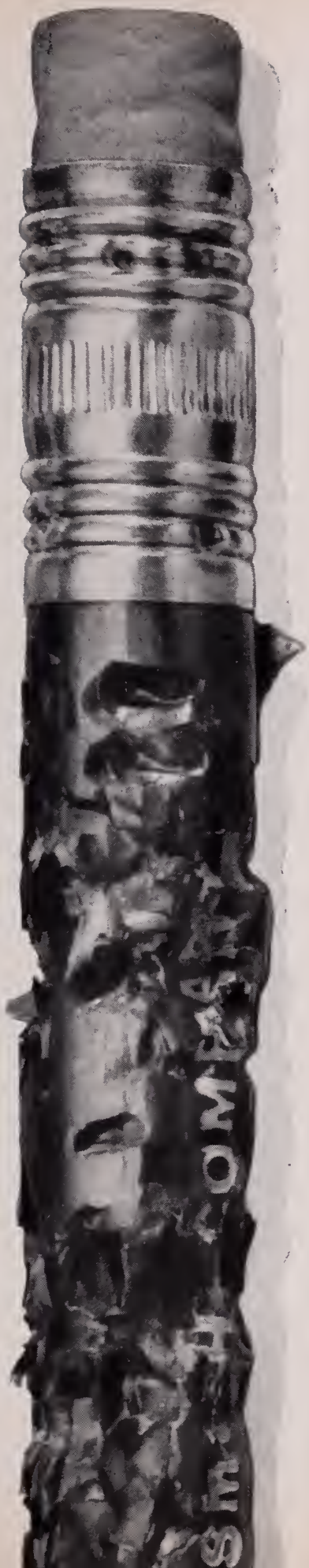
Today's regimented education, which places memory power above thinking power, would probably not have challenged Lincoln's hungry mind, nor have produced an individualistic thinker of his caliber.

Would he try to better himself and the land in which he lived, or would he have turned his considerable talents toward becoming a vociferous drone who expended his energy to achieve an increasingly larger dole from the public udder?

Lincoln's trademark is that of an uncouth, backwoods lad who came to be the savior of the Union and the symbol of pioneer spirit and American Democracy. Let's not relegate his spirit to a vaguely-associated national holiday, but employ his memory as a daily yardstick to measure the philosophical course of our government and our way of life!

What would our country be today without Honest Abe . . . what will the future hold with no more Lincolns?

Clyde T. Baker M.D.



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SEARLE

Special Articles

Factors Relating To Physician Distribution in Oklahoma 1950-1959

Part 1 of a Series

ROBERT C. LOWE, M.D.*
JAMES A. HAGANS, M.D.*
PEARL D. FISHER, Ph.D.*

THE CURRENT STUDIES of the health status and the medical care of the older age groups in the population cannot be accomplished without reference to the medical care resources available to the whole population. Of these resources, a competent and cohesive practicing physician population is necessary for adequate development, operation and function of all health facilities and personnel. It is essential that the medical profession prosper as its standards of service progress and the scope of its services broadens. This is necessary in order to attract others into the profession that it may reproduce itself and its quality at whatever rate is required by the demands for its services.

In what way do hospitals and city populations affect the location of physicians?

The trend of loss of physicians from the rural and smaller urban communities in Oklahoma described by Belcher[1] has continued. Associated with these net losses there has been a redistribution, the pattern of which is no doubt being infuenced by a number of factors. The extension of the quality and scope of service of which the profession is capable is logically a major influence. Population redistribution, supply and demand, community or regional economics, the geographic availability of hospital service of increasing quality and required complexity are some of the secondary and related forces currently at work to make the most effective use of the resources available.

Table I indicates that the net loss of physicians is inverse to community size. Between January 1, 1950 and January 1, 1959 only those communities with populations over 20,000** (1950) have, as a group, shown a net increase. On the other hand, of 204 communities with populations under 2500 (1950), 37 had no doctor of medicine in 1950. By 1959, 7 of these had acquired physicians but 53 more had lost theirs, (1-4) an increase to 83 (40.7% of the original group) without doctors of medicine. The evidence indicates that the end of this trend has not been reached.

The trouble with such statistical data, lacking information on offsetting trends and the rest of the picture, is that they may be interpreted offhand as deterioration in medical care. Such an interpretation does not consider the extent to which these changes are an expression of improvement brought

*All three of the authors are with the Department of Preventive Medicine and Public Health, University of Oklahoma School of Medicine; Doctors Hagans and Fisher are also with the Biostatistical Unit of the Department of P.M. and P.H.; in addition, Doctors Lowe and Hagans serve with the Department of Medicine.

**Excluding communities in Tulsa, Oklahoma and Muskogee counties.

about by other factors in the redistribution. It is evident that further information is needed. Table II shows the relationship of a hospital facility and community size to the changing pattern in communities with populations between 750 and 3500. Section A indicates that essentially the same proportion of physicians remained from 1950 to 1959 regardless of community size or the presence or absence of a hospital facility. It is worth noting, however, the tendency for communities with hospital facilities to retain fewer when the community size is small (i.e. less than 1500) than when it is larger (i.e. from 2500 to 3500).

Section B (table II) indicates that in small and intermediate size communities with hospitals a significantly greater proportion of new physicians was present in 1959 than was the case for the same size communities without hospitals. It should be noted that the proportion of new physicians in communities with hospitals decreased as the size of the community increased. The in-

(Continued on Page 113)

TABLE I

Pop. Range	No. of Towns	M.D.s 1950	M.D.s Remaining	M.D.s New	M.D.s 1959	% Change '50-'59
<750	106	97	48	11	59	-39.2
750-1499	61	94	39	24	63	-33.3
1500-2499	37	86	40	27	67	-22.1
2500-3499	20	86	47	24	71	-17.4
3500-4999	10	63	40	23	63	0.0
5000-9999	20	201	110	55	165	-17.9
10,000-14,999	6	80	53	17	70	-12.5
15,000-19,999	7	184	111	67	175	-4.9
20,000-36,000	6	163	115	71	189	+15.9

TABLE II

Pop. Range	without hospitals	with hospitals	statistical significance of difference
SECTION A: % of M.D.s present in 1950 and remaining in 1959—(chi square analysis*)			
750-1499	(33/74) 45%	(6/20) 30%	p > .05
1500-2499	(23/49) 47%	(17/27) 46%	p > .05
2500-3499	(9/20) 45%	(38/66) 58%	p > .05
Significance of Difference (vertically)	p > .05	p > .05	
SECTION B: % of total M.D.s in 1959 who were new since 1950—(chi square analysis*)			
750-1499	(13/46) 28%	(11/17) 65%	p < .01**
1500-2499	(8/31) 26%	(19/36) 53%	p < .02**
2500-3499	(7/16) 44%	(17/55) 31%	p > .05
Significance of Difference (vertically)	p > .05	p < .02**	
SECTION C: % change in total physicians 1950 to 1959—(chi square analysis*)			
750-1499	(28/74) -37.8%	(3/20) -15.0%	p < .05**
1500-2499	(18/49) -36.7%	(1/37) - 2.7%	p < .02**
2500-3499	(4/20) -20.0%	(11/66) -16.6%	p > .05
Significance of Difference (vertically)	p > .05	p > .05	

*p represents the probability that a difference as great or greater than that observed would have occurred by chance alone; when the value was .05 or less (1 in 20 or less) it was regarded as sufficiently rare as a chance occurrence to indicate a significant difference.

**indicates statistically significant difference.

News

TOP TALENT FEATURED FOR ANNUAL MEETING



ALLEN



DeBAKEY



DERBES



FELSON



KOHL



SKIBBINS



SOFIELD



VIRTUE

With twelve distinguished guest speakers confirmed, nearly one hundred high-quality scientific and technical exhibits scheduled, and an outstanding social program in the making, the Oklahoma State Medical Association's 1960 Annual Meeting promises to attract a record turnout. Oklahoma City will serve as host city this year, with all scientific events concentrated in the Municipal Auditorium and all social activities arranged at the Skirvin Tower Hotel. Dates for the Annual Meeting are May 2, 3, 4, 1960.

Under the leadership of General Chairman C. M. Bielstein, M.D., and Scientific Works Chairman Bert F. Keltz, M.D., the program has been completed at an early date. The unique composition of the Scientific Works Committee has contributed greatly toward the achievement of a diversified program of general interest. This year, the majority of specialty societies were represented by their respective presidents, as was the Oklahoma Chapter of the American Academy of General Practice.

Speakers Named

Listed as guest speakers for the event are: Edgar V. Allen, M.D., Internal Medicine, Rochester, Minnesota; Grayson Carroll, M.D., Urology, St. Louis, Missouri; Michael DeBakey, M.D., Surgery, Houston, Texas; Vincent J. Derbes, M.D., Dermatology, New Orleans, Louisiana; Eugene L. Derlacki, M.D., E.E.N.T., Chicago, Illinois; Murdina Desmond, M.D., Pediatrics, Houston, Texas; Benjamin Felson, M.D., Radiology, Cincinnati, Ohio; Schuyler Kohl, M.D., Obstetrics and Gynecology, Brooklyn, New York; Harold Rosen, M.D., Psychiatry, Baltimore, Maryland; Mr. Gerald J. Skibbins, Public Opinion, Princeton, New Jersey; Harold A. Sofield, M.D., Orthopedic Surgery, Oak Park, Illinois; and Robert W. Virtue, M.D., Anesthesiology, Denver, Colorado.

Public Opinion Expert Featured

Mr. Skibbins, Research Executive for the Opinion Research Corporation of Princeton, New Jersey, will provide an interesting di-

version from the scientific program. He will be the featured speaker at Tuesday's luncheon, where he will discuss public opinion surveys which his firm has conducted on behalf of the medical profession.

Panels Planned

The scientific program will be highlighted by two symposia-panel disucssion features.

On Tuesday morning, May 3, Doctors DeBakey, Allen and Virtue will present papers concerning current concepts of vascular disease. These formal presentations will be followed by a panel discussion and a question and answer period involving the three guest lecturers and a local pathologist.

The next morning, May 4, will find the entire concluding portion of the annual meeting devoted to rehabilitation, with particular emphasis on hemiplegia, spinal cord injuries, amputation and arthritis. This segment of the program will be under the direction of the Institute of Physical Medicine and Rehabilitation of NYU's Bellevue Medical Center. Local physical medicine personnel will cooperate in the presentation and will furnish demonstration subjects and equipment.

Social Highlights

Scientific activities will be adequately blended with social events in an effort to offer attenders a typically well-balanced meeting. Entertainment Committee members are now negotiating for a big-name orchestra to entertain at the President's Inaugural Dinner-Dance, which will be held in the Persian Room of the Skirvin Tower Hotel on Tuesday evening, May 3. The program will follow the usual pattern of dinner, installation ceremonies and dance.

President Alfred T. Baker, M.D., Durant, will culminate a busy year as the association's top executive officer when he presents the President's gavel to Walter E. Brown, M.D., Tulsa.

Entertainment Extra

Physicians and their wives who attend the meeting will be able to take advantage of additional topflight entertainment in the form of the Broadway production of "Look Homeward, Angel," which is scheduled at the Warner Theater on May 4 and 5. The Pulitzer Prize Winner, starring Miriam Hopkins, will be brought to Oklahoma City through the sponsorship of the Junior League.

Approximately two hundred tickets remain for each performance in price ranges of $5.75, $5.15, $3.75 (balcony) and $2.75 (balcony). The compactness of the theater should minimize the disadvantage of a rather limited selection of seats at this time.

Ticket orders should be accompanied by check and mailed to the Winn Ticket Agency, 135 West Main, Oklahoma City.

Oklahomans Participate In Texas Law-Science Week

Nine Oklahomans participated in Law-Science Week, held February 15-20 in Houston, Texas. Physician participants were: Earl D. McBride, M.D., William L. Waldrop, M.D., Louis J. West, M.D., and Robert A. Schneider, M.D., all of Oklahoma City.

The legal profession of the state was represented by Robert D. Looney, Fred M. Mock, and O. A. Cargill, Jr., of Oklahoma City, and Bryan W. Tabor, Tulsa. Mr. Ralph Snyder of Oklahoma City was also present.

Law-Science Week was organized by Hubert W. Smith, LL.B., M.D., Professor of Law and Legal Medicine, University of Texas, Director of the Institute and Chancellor of the Law-Science Academy. He was assisted by Warren P. McKenney, LL.B., Executive Assistant of the Institute and the Academy.

The six day program encompassed two short courses, directed to lawyers, physicians and claims men who are interested in medico-legal problems. The first short course, sponsored by the Law-Science Academy and Foundation was held February 15-17. The second, sponsored by the Law-Science Institute, was held February 17-20. Subject matter for both courses involved personal injury problems and medico-legal trial technique.

"Medicare" Expands Scope of Care

The Dependent's Medical Care Program has restored many of the benefits eliminated from the original program during the budgetary cutback of 1958. In October 1958, decreased appropriations rendered the Medicare Program financially unable to meet its obligations as originally established and agreed upon by state medical societies and the Department of Defense.

Flashback

Oklahoma had just completed re-negotiation of the contract for another fiscal year when the surprise announcement of the cutback was made at a special meeting in Washington, D.C. The abrupt about-face of the Federal government was followed, in turn, by a special meeting of the OSMA's House of Delegates, which promptly withdrew the association from any official sponsorship of the program and terminated all contractual arrangements. Blue Shield retained its position as fiscal agent on a month-to-month basis. Very basically, the restricted program slightly curtailed obstetrical benefits, eliminated all but emergency surgery and required military dependents who resided with their sponsors to first report to a military hospital for care. If facilities and/or personnel were found to be unavailable, the prospective patient would be issued a permit authorizing civilian care.

New Program Outlined

While the new program liberalizes the benefits to some extent, it is not a complete restoration of the original agreement.

Probably the most important *unchanged* feature of the program is the continued requirement of the permit system. The law states that spouses and children residing with their military sponsor are not eligible for civilian medical care and hospitalization privileges under Medicare *unless* the military medical facility nearest them is inadequate or unavailable. In such a case, a permit shall be issued by the Military Commander to qualify the eligible dependent to receive *authorized* civilian care at Federal expense. This permit must accompany the physician's claim, except in physician-certified emergencies. The details of this procedure remain unchanged and may be found on page two of the Medicare Manual.

The following types of care have been *restored* to authorized coverage:

1. Certain Surgery.
2. Acute emotional disorders of an emergency nature.
3. Laboratory and X-ray procedures preceding or following hospitalization *and* surgery.
4. Treatment of injuries on an out-patient basis.

Not restored, however, are neonatal visits and termination visits.

Surgery

Among the surgical procedures restored, are Stapes mobilization, eye surgery for glaucoma, cataracts and strabismus, and surgery for skeletal defects such as club foot. Payment for these procedures and the many others re-activated under the expanded program will be authorized only if the physician certifies that the surgery is for functional improvement or restoration.

Acute Emotional Disorders

Acute emotional disorders are now authorized and compensable if the condition constitutes a threat to the life or health of the patient. Twenty-one days coverage is the maximum allowed unless approved by prior arrangement.

Laboratory and X-Ray

Pre- and post-hospitalization tests and procedures, as authorized under the expanded program, do not include payment for outpatient office visits or physical examinations. They do include such tests and procedures when performed in or out of the hospital if the resulting surgery and hospitalization are authorized. Up to $75.00 is allowed pre-operatively and up to $50.00 post-operatively.

Out-Patient Treatment of Injuries

Treatment of bodily injuries on an outpatient basis is now authorized, but is lim-

ited to treatment of fractures, dislocations, lacerations and wounds. Compensation for such services will include payment for diagnostic and therapeutic tests and procedures as authorized by the physician.

The patient is still responsible for payment of the first $15.00 of the physician's total charges. Payment for laboratory tests, pathological and radiological examinations must not exceed $75.00.

Government Will Control and Interpret

Voluminous details concerning the expanded program will be interpreted by the Medicare Office of the Department of Defense. Neither the OSMA nor Blue Shield have jurisdiction in this area, although the latter will process claims as the fiscal agent.

Keogh Legislation Faces Crisis

The long-awaited Smathers-Morton-Keogh-Simpson Bill is now facing its most most crucial test in several sessions of Congress. It is now before the Senate Finance Committee in a make-it-or-break-it culmination of nine years' promotion by the medical profession and other self-employed groups.

Last year, the House of Representatives passed the measure and referred it to the Senate where it rested without action until congressional adjournment. Thus, the recommendation of the Senate Finance Committee will have great influence on its final disposition.

OSMA Action

Under the direction of J. R. Stacy, M.D., the association's Federal Legislative Committee has been conducting a write-in campaign among the membership, as have other interested associations and groups. The letters are being directed to Senator Robert S. Kerr, Senate Office Building, Washington, D.C. Senator Kerr is the second-ranking member of the Finance Committee and will undoubtedly have much to say about the committee's recommendation. Although the senator is aware of the medical profession's general attitude toward this legislation, a display of unity at this time would assist him in properly representing the self-employed of his state. County medical societies and individual physicians are encouraged to write.

Tax Equity for the Self-Employed

The purpose of the bill is to permit a self-employed person to defer income tax each year on $2500 or 10% of income, whichever is less, but not to exceed $50,000 during a life-time. For those who have reached the age of fifty prior to the effective date, provision is made to allow additional amounts to be set aside in order to build up an adequate fund during productive years.

Under the bill, a person may claim a deduction for an investment in (1) a restricted retirement policy (life, endowment or annuity; but deductions are not allowed for costs allowable to the life insurance protection); or, (2) a restricted retirement fund trusteed with a bank.

Why Is It Necessary?

High taxes and inflated living costs make it difficult for the self-employed person to set aside money for retirement, in the absence of tax deferments which are already available to corporation employees. In many instances, qualified young men are going on a payroll rather than striking out for themselves as enterpreneurs. This legislation would help counteract this shift away from individual enterprise.

About ten million persons are now penalized by the tax laws. The Old-Age and Survivors Insurance program is intended to provide the minimum benefits necessary for a subsistence standard of living, and self-employed persons should have the right, as do salaried employees, of providing a second layer of income for retirement and survivorship's benefits. This is even more desirable and equitable in the case of physicians, who have elected to abstain from participation in the Social Security scheme.

If the bill passes, physicians will not pay taxes on the set-aside until it is withdrawn from the selected fund, thus paying a lesser tax rate during less-productive years.

Third Oklahoma Colloquy Planned



DAVID M. HUME, M.D.

Use of the adrenal steroids in surgical cases, in collagen disease and in allergic problems will be among the clinical aspects covered by guest lecturers at the Third Oklahoma Colloquy on Advances in Medicine March 24-26 at the University of Oklahoma Medical Center.

David M. Hume, M.D., chairman, Department of Surgery, Medical College of Virginia, Richmond, will discuss use of the steroids in the surgical patient at a sectional dinner meeting sponsored by the Oklahoma City Surgical Society March 25 at Glen's Hick'ry Inn.

In addition, Doctor Hume will address a general session March 24 on "The Mechanisms Controlling Adrenal Cortical Secretion in Response to Trauma." Doctor Hume has published extensively in the areas of adrenal cortical secretion and of renal and other tissue transplantation.

Bram Rose, M.D., Ph.D., associate professor of medicine, McGill University, will appear before both morning and afternoon general sessions March 26. He will talk on the use of anti-inflammatory steroids in the control of collagen diseases and in the management of allergy.

President-elect of the American Academy of Allergy, Doctor Rose is on the editorial boards of the Journal of Allergy and the International Archives of Allergy and Immunology. His most recent research has



BRAM ROSE, M.D., Ph.D



LAWSON WILKINS, M.D.

been carried out on antigen-antibody mechanisms with particular reference to those related to the common forms of allergy.

Lectures by Lawson Wilkins, M.D., professor of pediatrics, Johns Hopkins University School of Medicine, are scheduled at the morning session March 25 and at an afternoon section meeting sponsored by the Oklahoma City Pediatrics Society the same day.

Doctor Wilkins' studies have helped clarify the problems of abnormal sex differentiation and gonadal dysgenesis. He is author of "The Diagnosis and Treatment of Endocrine Disorders in Childhood and Adolescence."

His general session lecture is entitled "Diagnosis and Treatment of the Adrenogenital Syndrome." At the Pediatrics session, he will speak on "Abnormalities of Sex Differentiation: Etiology, Diagnosis, Selection of Sex and Treatment."

The three are among 15 guest speakers and 13 members of the Medical Center faculty who will present papers during the conference on adrenal steroids. Special interest meetings also are planned in the fields of medicine and basic science.

Oklahomans Attend Blue Shield Conference

More than three hundred delegates, including state and local medical society officers and secretaries, as well as Blue Shield Plan physician-trustees and executives, attended the 1960 Blue Shield Professional Relations Conference which was held February 1-3 at the Drake Hotel in Chicago. President A. T. Baker, M.D., and Prepaid Medical Care Committee members, Carl Lindstrom, M.D., and R. G. Obermiller, M.D., represented Oklahoma.

The Professional Relations Conference program was prepared under the direction of the National Association's Professional Relations Committee and had as its theme this year, "Facing the Facts—In the Future of Blue Shield."

"The new year presents a serious challenge to Blue Shield and to the entire voluntary health care movement," the advance publicity stated, "for the simple reason that it is an election year and we can expect a supreme effort by the backers of Forand-type legislation to get their program passed in Congress. To give our physician delegates as much of an up-to-date picture as possible, we have scheduled discussions on federal legislative activities and on Blue Shield activities with regard to the development of programs for senior citizens.

Among the speakers were Doctor John Reckless of Duke University, who discussed "The Facts of Life in the Future of American Medicine;" Doctor Charles G. Hayden, Executive Director of the Massachusetts Blue Shield Plan, who spoke on "Evolving New Benefit Patterns to Meet the Demands for Expanded Forms of Coverage—An Examination of Factors of Interest and Importance to the Profession;" and Doctor Ira C. Layton, of Kansas City, Missouri, who talked about "The Dynamics of Blue Shield and Their Relation to the Future of Private Medical Care." Also on the program were Thomas M. Newell, Director, National Field Service, New York City, whose subject was "Public Opinion and Its Application in Shaping Future Developments in Blue Shield," and Doctor Donald Stubbs, Chairman of the

Board of the National Association of Blue Shield Plans, who talked about "The Plan Governing Board—An Examination of Concepts Affecting Its Composition Functions and Responsibilities."

AAGP Will Hold 12th Annual Scientific Assembly

The 12th Annual Scientific Assembly of the American Academy of General Practice will be held in Philadelphia's Convention Hall, March 21-24, 1960. Approximately 4,000 family doctors and 3,000 residents, interns, exhibitors and wives will attend the four-day program which will highlight recent progress in medicine and surgery.

The Philadelphia Assembly program lists 31 prominent medical educators who will discuss a variety of subjects ranging from arthritis and anemia to surgery, geriatrics and mental health. The doctors will also visit more than 100 scientific and 300 technical exhibits.

During the meeting, the doctors will have a chance to be on the other end of a stethoscope during physical examination sessions. More than 600 doctors took physical examinations at the last Academy meeting. Many learned, for the first time, that they needed immediate medical attention.

Fifty Year Club to Be Organized Within AMA

J. H. McCurry, M.D., of Cash, Arkansas, advises that he has the approval of the American Medical Association to organize a Fifty Year Club within the AMA. Doctor McCurry is anxious to hear from physicians who have been in practice fifty years or more who desire to become members of this club, giving their name and a complete address.

The first meeting is to be held in Washington, D.C. at the Clinical meeting November 29 to December 2, 1960.

OB-GYN Exams Scheduled

The American Board of Obstetrics and Gynecology has scheduled examinations (Part II), oral and clinical for all candidates from April 11 through 16, 1960 at the Edgewater Beach Hotel, Chicago, Illinois.

Medical Center Presents Service Awards



W. K. West, M.D., left, and Dean Mark R. Everett at annual service award ceremonies at the University of Oklahoma Medical Center. Doctor West received a 40-year pin.

Sixty-one faculty members, including 11 preceptors, were cited at the University of Oklahoma Medical Center's fourth annual service awards program December 10.

Heading the list was W. K. West, M.D., professor of orthopedic surgery, honored for serving 40 years on the School of Medicine faculty.

Pins were awarded by Mark R. Everett, Ph.D., dean and director of the Center, who was presented a 35-year citation by Lloyd E. Swearingen, Ph.D., vice-president in charge of research and development at the University of Oklahoma, Norman.

Other 35-year pins went to: James B. Eskridge Jr., M.D., professor of obstetrics; Leroy Long, M.D., clinical professor of surgery; Joseph C. MacDonald, M.D., professor of otorhinolaryngology; Leo Starry, M.D., professor of surgery.

Thirty year awards were given: Louis H. Charney, M.D., associate professor of medicine; Ephraim Goldfain, M.D., assistant professor of medicine; R. Q. Goodwin, M.D., professor of medicine; W. Jeanne Green, Ph.D., assistant professor of pathology; Joseph W. Kelso, M.D., professor of gynecology; Lawrence C. McHenry, M.D., pro-

fessor of otorhinolaryngology; James F. Moorman, M.D., associate professor of medicine; Charles R. Rountree, M.D., clinical professor of orthopedic surgery; James B. Snow, M.D., professor of pediatrics.

Ernest Lachman, Ph.D., professor and chairman of the Department of Anatomy, received a 25-year pin.

Those who reached the 20-year milestone were: Meredith M. Appleton, M.D., associate professor of urology; Robert B. Howard, M.D., clinical professor of surgery; Harry L. Deupree, M.D., associate professor of obstetrics; Grady F. Mathews, M.D., professor of preventive medicine and public health; Newman S. Matthews, M.D., assistant professor of medicine; Charles M. O'Leary, M.D., clinical professor of surgery; Moorman P. Prosser, M.D., clinical professor of psychiatry; Charles D. Tool, M.D., assistant professor of pathology; Robert I. Trent, M.D., clinical professor of ophthalmology.

Other awards to members of the faculty in Oklahoma City:

Fifteen years—N. F. Barkett, M.D., associate professor of surgery; Robert H. Bayley, M.D., professor of medicine; Florene C. Kelly, Ph.D., professor of microbiology.

Ten years—James C. Amspacher, M.D., assistant professor of orthopedic surgery; C. Alton Brown, M.D., associate professor of medicine; James R. Colvert, M.D., assistant professor of medicine; Sterling T. Crawford, M.D., assistant professor of gynecology; John J. Donnell, M.D., associate professor of medicine; Louis S. Frank, M.D., assistant professor of pediatrics; Walter S. Hendren, M.D., assistant professor of medicine; Robert G. Hirschi, M.D., assistant professor of oral surgery; Ella M. George, M.D., assoriate professor of physical medicine; George H. Guthrey, M.D.. associate professor of psychiatry; Robert P. Holt, M.D., assistant professor of orthopedic surgery; Dick H. Huff, M.D., assistant professor of medicine; Walter Joel, M.D., professor of pathology; Robert W. Kahn, M.D., assistant professor of medicine; A. C. Lisle, M.D., assistant professor of surgery; William G. McCreight, M.D., assistant professor of dermatology; Robert McKee, M.D., assistant professor of obstetrics; Milton J. Serwer, M.D., professor of obstetrics; Harold Sleeper. M.D., assistant professor of psychiatry; Carl T. Steen, M.D., innstructor in psychiatry; L. Duncan Thelkeld, M.D., associate professor of obstetrics; William L. Waldrop, M.D., assistant professor of orthopedic surgery; George L. Winn, M.D., assistant professor of medicine.

The Medical Center this year started awarding service pins to preceptors who have served five or ten years. First awards were made to the following:

Ten years—Carl Bailey, M.D., Stroud; W. C. McCurdy, M.D., Purcell; E. A. McGrew, M.D., Beaver; Joe L. Duer, M.D., Woodward.

Five years—Lynn C. Barnes, M.D., Nowata; Walter H. Dersch Jr., Shattuck; George Gathers, M.D., Stillwater; Robert B. Gibson, M.D., Ponca City; Kenneth Godfrey, M.D., Okeene; Edward T. Shirley, M.D., Wynnewood; Carlton E. Smith, M.D., Henryetta.

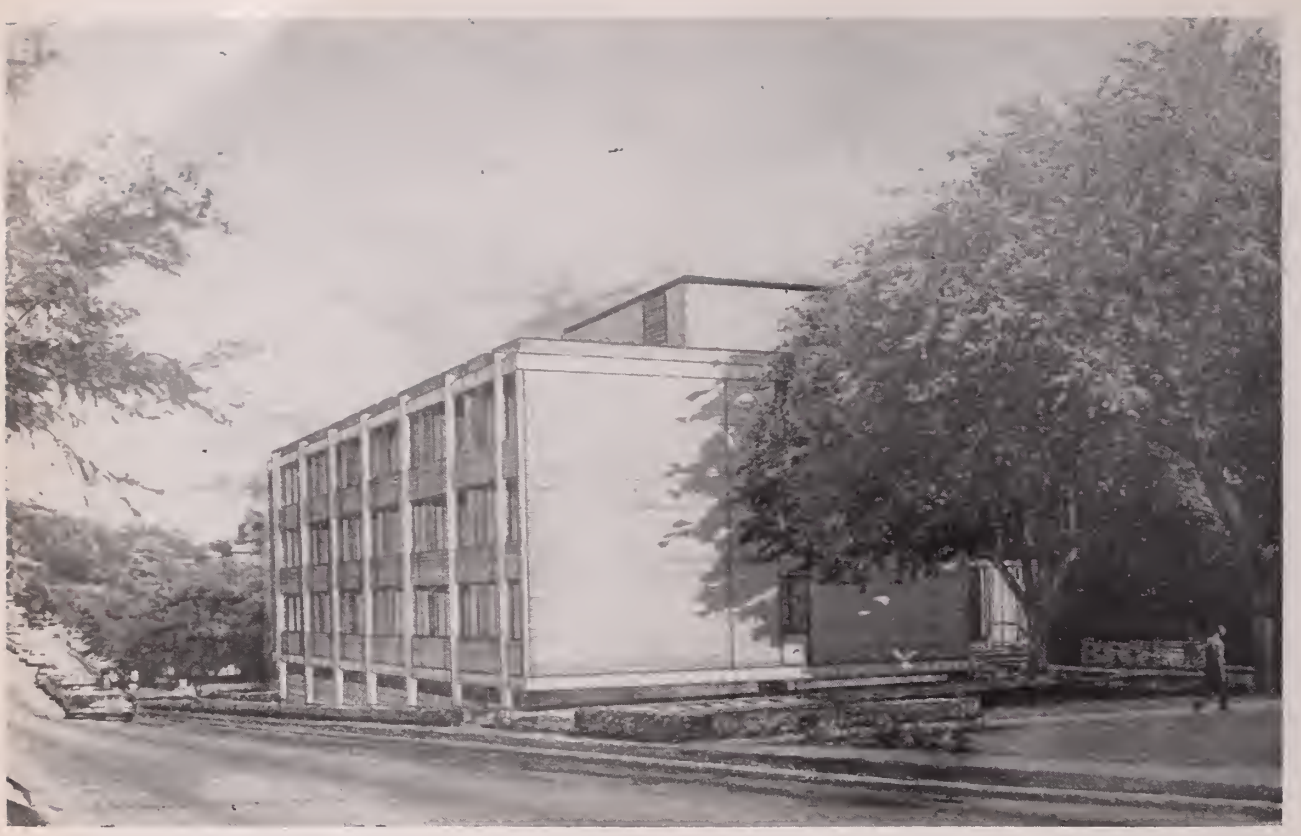
PHYSICIAN DISTRIBUTION

(Continued from Page 105)

verse relationship between this trend and that for physicians remaining from 1950 to 1959 in these same communities (Section A) suggests the explanation for these findings, i.e. the smaller the proportion remaining, the larger the new physician proportion is likely to be under these circumstances.

Section C (table II) demonstrates that the small and intermediate size communities with hospitals showed a significantly lower net loss of physicians, compared to their counterparts without hospitals. The lack of significant difference between communities of 2500 to 3500 population, with and without hospitals, points up the question as to whether or not the presence of a hospital per se is the primary factor influencing changes in physician population.

(To Be Continued)



Pictured above is the $800,000 expansion of the Oklahoma Medical Research Foundation. The building will be be constructed on the University Hóspital property.

OU Medical Research Building Under Way

Groundbreaking ceremonies for the University of Oklahoma Medical Research Building were held January 5 under joint sponsorship of the Center and the Oklahoma City Chamber of Commerce.

Paul A. Weiss, Ph.D., head of the Laboratory of Developmental Biology at the Rockefeller Institute for Medical Research, New York City, was principal speaker, addressing a luncheon at the Biltmore Hotel and speaking again at rites in the School of Medicine Auditorium. He came here as guest of Frontiers of Science Foundation.

Snowfall forced the actual groundbreaking indoors, where dignitaries performed the symbolic shoveling at a long wooden box filled with soil from the site. Bryce Baggett, Oklahoma City, state representative, presided at the brief program.

The approximately $800,000 building is being constructed on the south side of NE 13 Street in front of University Hospital and to the west of the hospital's Outpatient Clinics wing. The four-story structure will house some 50 laboratories and related facilities. University of Oklahoma Regents let the contract to Builders Construction Company of Oklahoma City at their December meeting.

Construction of much-needed laboratory space for faculty members was made possible by a $400,000 federal health research facilities grant awarded in 1957 and matched by a $400,000 appropriation of the 1959 state legislature.

Other speakers at the double-barrelled program included Lieut. Gov. George Nigh, George L. Cross, Ph.D., University of Oklahoma president, and Mark R. Everett, Ph.D., dean and director of the Center. Truett Coston, architect, was master of ceremonies at the luncheon.



Front view of Tinker's $2,368,962 modern hospital and dental clinic which was dedicated Saturday, January 23 by Dudley C. Sharp, Secretary of the Air Force.

New Tinker Air Force Base Hospital Opened

Formal dedication ceremonies were held January 23 for the new Tinker Air Force Base Hospital in Oklahoma City. Principle speaker for the occasion was Dudley C. Sharp, Secretary of the Air Force.

Covering 11 acres of the new area donated to the Air Force by the Oklahoma City Chamber of Commerce, the hospital is the first Air Force-designed hospital and dental clinic in the Air Force Medical Service.

The 75-bed fireproof hospital, with a 150-bed expansible plan, was built at a cost of $2,368,962. Construction was begun in May 1958 and completed in November 1959. It features complete air conditioning, with a thermostat in every room. There is piped-in central oxygen and vacuum to each patient area, a music distribution system with outlets to each patient's bed and nurses radio communication system to each patient. All patient bedrooms are semi-private with private baths and there are telephone and television outlets by each bed.

The east wing of the hospital houses the dental clinic which contains six dental operating rooms and two oral surgery rooms.

Three major operating rooms, an orthopedic plaster and splint room, sterile supply room and a post-surgical recovery room comprise the surgical suite.

Offices for the director, base medical services and the executive are located in the administrative wing. Also included in this wing are the business office, medical library and staff conference room.

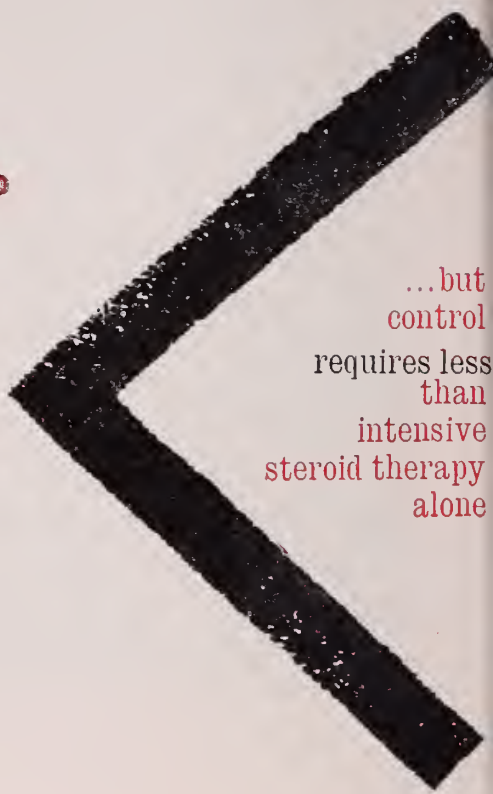
The modern structure, with its terrazo, asphalt and vinyl asbestos tile floors, mosaic ceramic-walled lobby, modern coved lighting, hi-fi music speakers, acoustical ceilings and hospital exchange is a valuable addition to the permanent Air Force facilities at Tinker Air Force Base.

Architects for the structure were Coston, Frankfurt and Short, Oklahoma City.



when the
rheumatic
disorder
is more
than salicylates
alone
can control...

MORE
HIGHLY INDIVIDUALIZED
THERAPY
FOR THE
RHEUMATIC
"IN-BETWEEN"



...but
control
requires less
than
intensive
steroid therapy
alone

Aristo

wider latitude in adjusting dosage

ARISTOGESIC is particularly effective for relief of chronic — but less severe — pain of rheumatic origin. ARISTOGESIC combines the anti-inflammatory effects of ARISTOCORT® Triamcinolone with the analgesic action of salicylamide, a highly potent salicylate. Dosage requirements for ARISTOGESIC are substantially lower than generally required for each agent alone. The exceptionally wide latitude of dosage adjustment with ARISTOGESIC permits well-tolerated therapy for long periods of time with fewer side effects.

Indications: Mild cases of rheumatoid arthritis, tenosynovitis, synovitis, bursitis, mild spondylitis, myositis, fibrositis, neuritis, and certain muscular strains.

Dosage: Average initial dosage: 2 capsules 3 or 4 times daily. Maintenance dosage to be adjusted according to response.

Precautions: All precautions and contraindications traditional to corticosteroid therapy should be observed. The amount of drug used should be carefully adjusted to the lowest dosage which will suppress symptoms. Discontinuance of therapy must be carried out gradually after patients have been on steroids for prolonged periods.

Each ARISTOGESIC Capsule contains:

ARISTOCORT® Triamcinolone	0.5 mg.
Salicylamide	325 mg.
Dried Aluminum Hydroxide Gel	75 mg.
Ascorbic Acid	20 mg.

Supply: Bottles of 100 and 1,000.



Steroid-Analgesic Compound LEDERLE

Lederle

LE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, New York



Have something to exhibit

If so, you should apply now for space at the 1960 meeting of your association. Scientific Exhibits will again play a major role . . . so why not prepare a graphic presentation of your work and share it with the 700 physicians who will attend the state's largest medical meeting? A research or clinical project may be shown as an individual exhibit, or you may present a single case in the "Klinical Kaleidoscope" section.

Research or clinical project

Perhaps you have done some original research, undertaken some experimental work, or simply drawn some conclusions based upon a series of interesting cases. Then you would be eligible to apply for an exhibit booth of your own in the Scientific Exhibit Section. A subject which has a special visual interest would be particularly suitable . . . and it need not be large—just stimulating!

Bizarre case

All of us have encountered a unique case which is worth talking about. If you have a well-documented one which you would like to share with others, toss your hat in the ring today by completing the application on the opposite page. The "Klinical Kaleidoscope" section will be a varied selection of bizarre cases, each presented with one to five x-rays or illustrations and accompanied by a very brief abstract of the clinical course.

O.S.M.A. ANNUAL MEETING

municipal auditorium · oklahoma city

may 2·3·4, 1960

APPLICATION FOR SCIENTIFIC EXHIBIT SPACE

54th Annual Meeting

OKLAHOMA STATE MEDICAL ASSOCIATION

Municipal Auditorium — Oklahoma City — May 2, 3, 4, 1960

1. NAME AND ADDRESS OF APPLICANT:

2. TITLE OF EXHIBIT:

3. INDIVIDUAL BOOTH? ☐ "KLINICAL KALEIDOSCOPE" SECTION? ☐

4. SPACE REQUIRED:

   A. BOOTH: Indicate width in multiples of eight feet _____
   (All booths will be 6 feet in depth)

   B. "KLINICAL KALEIDOSCOPE": Indicate square footage:
   Wall-type display _____ Table top _____

5. A. GENERAL DESCRIPTION:

   B. MATERIAL TO BE SHOWN (Subject matter):

   C. METHOD OF PRESENTATION (x-ray, photos, graphs, models, etc.):

6. PREVIOUS SHOWINGS

ACT NOW — SPACE IS LIMITED

Applications Must Be Received Prior to March 1, 1960

Oklahoma State Medical Association • Box 9696 • Oklahoma City



Linkletter Honors GP of the Year

The American Medical Association's GP of the Year, Chesley M. Martin, M.D., Elgin, Oklahoma appeared as a special guest on Art Linkletter's House Party, December 4, CBS-TV. Pictured above are the Martins with their host Mr. Linkletter.

What's Your Hobby, Doctor?

The DOCTOR'S HOBBY SHOW has become one of the outstanding attractions at the OSMA ANNUAL MEETING. A project of the Woman's Auxiliary, the show offers physicians an excellent opportunity to display the products of their leisure time. If you have a hobby, don't keep it a secret . . . Show your colleagues what you can do . . . APPLY NOW!

Doctor's Hobby Show

O.S.M.A. Annual Meeting

Municipal Auditorium

Oklahoma City

MAY 2, 3, 4, 1960

Application For Hobby Show Space

54th ANNUAL MEETING

OKLAHOMA STATE MEDICAL ASSOCIATION

DESCRIBE EXHIBIT, including information as to size, shape and value (insurance is provided):

IMPORTANT: Deliver Exhibit to Zebra Room, Municipal Auditorium by noon, May 1. Your Exhibit will be personally attended and insured at all times. It must be picked up by noon, May 4, when management responsibility ends.

MAIL THIS FORM TODAY!

State Chairman—Mrs. William R. R. Loney
2440 E. 26th Place
Tulsa 14, Oklahoma

State Co-Chairman—Mrs. Harrell C. Dodson, Jr.
1108 N.E. 42nd Street
Oklahoma City, Oklahoma

Course on Cardiac Patient Held

Four guest lecturers participated in a postgraduate short course on rehabilitation of the cardiac patient sponsored by the Oklahoma City Internist Association and Oklahoma State Heart Association February 10 at the University of Oklahoma Medical Center.

They were: Carlton B. Chapman. M.D., professor of medicine, Southwestern Medical School, Dallas; Amasa B. Ford, M.D., director of Rehabilitation Teaching Program, Western Reserve University, Cleveland; L. E. January, M.D., professor of medicine, State University of Iowa, Iowa City; Frederick J. Kottke, M.D., professor and head of the Department of Physical Medicine and Rehabilitation, University of Minnesota Medical School, Minneapolis.

The short course was combined with the Internist Association's annual Washington Birthday Clinic.

Doctor Chapman spoke on cardiovascular response to exercise and gave a second lecture on exercise in patients with mitral valve disease. Doctor Ford discussed various aspects of the relationship of physical activity to heart disease.

Problems in management of agricultural workers with heart disease were outlined by Doctor January. Doctor Kottke's subjects were "Cardiac Demands of Hospital and Everyday Activities" and "Planning a Rehabilitation Program for the Cardiac Patient."

Sessions began at 1 p.m. and 7 p.m. in the Medical School Auditorium. Seven faculty members took part. Guest participants included Judge Marx Childers, presiding judge, Oklahoma State Industrial Commission; Camp S. Huntington, M.D., assistant medical director, Phillips Petroleum Co.; Voyle Scurlock, director, Division of Vocational Rehabilitation, State Board of Education; and C. S. Lewis Jr., M.D., Tulsa, president, Oklahoma State Heart Association.

25 YEARS AGO



Articles published in *The Journal* of the Oklahoma State Medical Association, February, 1935.

DIABETIC PROBLEMS

Lea A. Reily, M.D.
Professor of Clinical Medicine, University of Oklahoma
Oklahoma City

Sir William Gull, Queen Victoria's physician, said: "What sin has Pavy committed or his father before him that he should be condemned to spend his life seeking a cure for an incurable disease?" Thus now we are sitting almost within the shadow of the laboratory where a definite treatment has been found lengthening the span of life so afflicted. This has been the outstanding development in medicine for the last decade. That, together with the fact that we are saving our children from the acute sudden deaths and condemning them to old age and diabetes. The high nervous strain of modern life with the excessive eating, has brought diabetes to morbidity and mortality. It is estimated in the states that there are one million diabetics, and that the mortality has raised from 20th to 10th place as the cause of death.

Mark Twain facetiously put it, "The way to attain longevity is to have a chronic disease and continually treat it." Joslin dedicated his 1934 Manual "To those individuals who have conquered diabetes by living longer with it than they were expected to live without it." Earlier diagnosis and better treatment as in tuberculosis, result in cases of longer duration and in compliance with the above, extends life and gives more chances for complications. The present diabetic is half and half a medical and surgical diabetic. Surgery is becoming more and more an increasing object of importance in handling those so afflicted, and the surgeon is faced with the care of a group of cases requiring special consideration, and leans on the internist for his advice and cooperation.

So we have direct and indirect complications in diabetes which touch all specialties in medicine, but I would liken the relationship of John Saxes' poem, where he described the Seven Blind Men of Hindoostan, who were wo wondrous wise, who went to see the elephant although none of them had eyes. Each saw the elephant from his own viewpoint, each one had a perception, but it takes conception of the diabetic in the form of some one physician who is in complete charge of the case in a medical ward who treats the diabetes, acting as the brains of the elephant who not only sees and governs the body as a whole, but who is not unmindful of the different members, remembering that diabetes is as jealous of her stellar role as an opera singer, and must be handled as carefully.

Letters

Dear Doctor Payne:

I noted with interest in this last issue of Round-up mention of your co-authorship of a paper on Clinical Evaluation of Weak Analgesic Agents. This brings to my attention again some recent thoughts I have had about analgesics and also muscle relaxing drugs which are put on the market in large amounts with a good deal of high pressure salesmanship on the part of the pharmaceutical firms. I note that in the last few years there has been an increasing tendency to have these drugs tested on what often seems to be a highly uncritical basis by investigators who are frequently not working in a University or comparable medical center. The investigation then is written up and published in one or another of the innumerable medical journals and reprints of the articles are used as sales promotion material by the drug firms. I strongly feel that in evaluating medications where there is so much difficulty in offering clear-cut objective evidence of effect, or lack thereof, the double-blind method of testing should routinely be employed and little attention paid to results obtained in any other way. Of course, it is quite difficult to offer any method of controlling this flood of questionably scientific literature, but it does seem to me at least that the editors of journals should be urged to take a much more closely critical look at papers submitted to them for publication. Possibly the Medical Writers' Society would be a place where someone could sound off on this subject and perhaps thereby bring it to the attention of editors of some of the journals.

I am taking the liberty of sending a copy of this letter to Dr. Ben Nicholson since the idea I mention may possibly be of interest to him.

I would like very much to see a copy of your paper on analgesic agents whenever it is conveniently available.

Sincerely,
S. FULTON TOMPKINS, M.D.

Dr. Tompkin's criticism is well taken and we state journal editors have little defense. In justice to us, however, it must be understood that our job as editors is not just selecting papers for publication from a great number that are submitted but procuring papers to make a decent journal since very few are submitted. In addition, we have a responsibility to our membership and the J. O. S. M. A. has adopted a policy that if a member submits a paper it will be accepted unless completely hopeless. We will not, in anyway, discourage a member who wants to write or report his work. Our greatest sin is the granting of permission to obtain large numbers of reprints for promotional purposes. In this we have no defense nor should we try to develop one.—The Editors.

Dear Doctor Nicholson:

The Oklahoma Society of Anesthesiologists wishes to announce the establishment of a Clinical Anesthesia Study Committee. This committee is set up to serve members of our Society and the Oklahoma State Medical Association by reviewing anesthesia protocols submitted by any Surgeon or Anesthetist.

The Committee is composed of four Anesthesiologists each of whom will review the protocol and make comments and recommendations which will be forwarded to the person submitting the case.

An elaborate procedure has been set up to assure complete anonymity of the patient,

surgeon, and person administering the anesthesia in every case. The Secretary of the Oklahoma Society will be the only person knowing both the name of the person submitting a case and its number, and he will never see either the case report or the comments.

The Committee solicits cases for study from surgeons or their anesthetists in addition to the members of our Anesthesia Society. Blanks for submitting case reports may be obtained free by writing to the Secretary of the Oklahoma Society of Anesthesiologists, c/o Department of Anesthesia, University Medical Center, Oklahoma City, Oklahoma. The Committee reserves the right to publish interesting cases without identifying numbers or names in this *Journal* or other medical journals. Cases cannot be reviewed unless they are reported on the standard forms furnished by our Society.

Sincerely yours,
R. GIBSON PARRISH, M.D.

The Journal will publish the reviews that are submitted by the committee.

Have You Heard?

ROBERT H. FURMAN, M.D., director of cardiovascular research at the Oklahoma Medical Research Foundation, has been appointed to the heart study section of the National Institutes of Health, research arm of the U.S. Public Health Department. Members of the study section are investigators who review all applications for grants from the public health service to support research in heart disease.

WILLIAM W. SCHOTTSTAEDT, M.D., University of Oklahoma Medical Center faculty member since 1953, has been named chairman of the Department of Preventive Medicine and Public Health. He succeeds Kirk T. Mosley, M.D., associate dean in charge of special training and research programs.

LEO LOWBEER, M.D., pathologist at Hillcrest Medical Center, Tulsa, will be a lecturer at the 12th Annual Meeting of the American Academy of Forensic Sciences, March 3-5, in Chicago.

EVANS JAMES KURTS, M.D., is joining A. W. Haddox, M.D., as a partner in the Haddox Clinic in Antlers. Doctor Kurts is a graduate of the College of Medicine, University of Tennessee, Memphis, and has been taking a residency in surgery at Hillcrest Hospital in Tulsa.

C. A. TRAVERSE, M.D., Alva, has instigated a rather unique feature at an Alva hospital staff meeting. ART YOUNG, M.D., orthopedic specialist from Freeport, New York, discussed practical orthopedic problems with the group for one hour and fifteen minutes via amateur "ham" radio. The lecture proved so interesting and profitable that a similar "meeting" of the specialist and Alva physicians has been planned for a later date.

LYNN BARNES, M.D., Nowata physician, has been named local President of the Chamber of Commerce for 1960.

CHARLES W. LETCHER, M.D., Miami, retired from the Navy, has received a commission as Rear Admiral (Medical Corps) United States Naval Reserve.

ROBERT K. MCINTOSH, M.D., has been selected Outstanding Citizen of 1959 in Tahlequah. While most letters of recommendation for the honor mentioned a single area of service, one seemed to sum up the appreciation " . . . Doctor McIntosh has given his life to this community, commanding the highest respect in his profession. Tahlequah is lucky to have doctors like him."

Coming Meetings

UNIVERSITY OF OKLAHOMA MEDICAL CENTER
POSTGRADUATE PROGRAM*
Individual Postgraduate Courses

BASIC ELECTROCARDIOGRAPHY — February 29 through March 5.

OPTHALMOLOGY-OTOLARYNGOLOGY SYMPOSIUM—March 3 and 4.

OBSTETRICS-GYNECOLOGY SYMPOSIUM—March 5.

ADRENAL STEROIDS—March 24, 25, and 26. (Third Oklahoma Colloquy on advances in Medicine.)

ORTHOPEDIC SYMPOSIUM—May 13 and 14.

CARCINOMA OF THE SKIN—April 22 and 23. (Sixth Annual Combined Surgery, Radiology, Pathology Symposium.)

OKLAHOMA ASSSOCIATION OF HOUSE STAFF PHYSICIANS—May 6.

PEDIATRIC NEUROLOGY—May 12 and 13. This symposium to be held in conjunction with the Wednesday Short Course on May 11.

SERIAL POSTGRADUATE COURSE

Postgraduate Division

Oklahoma City, Oklahoma
1959-1960

Wednesday Short Courses
3:30 to 8:30 p.m.

Mar. 9	C. B. Taylor Lectureship and Urology Symposium—Guest Lecturer to be announced
Apr. 13	Anesthesia for the Part-time Anesthetist
May 11	Neurological Diseases in Childhood
June 8	Surgery

*Further information may be obtained from the Office of Postgraduate Education, University of Oklahoma School of Medicine, 801 N.E. 13th Street, Oklahoma City, Oklahoma.

OKLAHOMA CHAPTER

AMERICAN ACADEMY OF GENERAL PRACTICE

February 15-16, 1960 **Tulsa Hotel**

Tulsa, Oklahoma

The 12th Annual Meeting of the Oklahoma Chapter of the American Academy of General Practice will be held at the Tulsa Hotel, February 15-16, 1960, in Tulsa. Complete program on page 43.

UNIVERSITY OF TEXAS
POSTGRADUATE SCHOOL OF MEDICINE

February 25, 26, 27, 1960 **Houston 25, Texas**

The University of Texas Postgraduate School of Medicine is presenting a program on Gastroenterology in the Texas Medical Center, Houston, Texas on February 25, 26, 27, 1960. Emphasis on diagnosis and therapeutic approaches, medically and surgically, will be discussed.

For further information write: Office of the Dean, The University of Texas Postgraduate School of Medicine, 410 Jesse Jones Library Building, Texas Medical Center, Houston 25, Texas.

TEXAS POSTGRADUATE SCHOOL OF MEDICINE

Fifth Annual Course in Anesthesiology

March 9, 10 ,11, 1960 **Houston, Texas**

The University of Texas Postgraduate School of Medicine announces the Fifth Annual Course in Anesthesiology to be held March 9, 10, 11, 1960 in Houston. The course is designed to review theory and practice of commonly used anesthetic techniques. Inquiries may be addressed to The University of Texas, Postgraduate School of Medicine, 410 Jesse Jones Library Building, Texas Medical Center, Houston 25, Texas.

SCOTT AND WHITE CLINIC

EIGHTH ANNUAL POSTGRADUATE CONFERENCE

March 6, 7, 8, 1960 **Temple, Texas**

This course, Current Topics in Medicine and Surgery, will be presented by Scott, Sherwood and Brindley Foundation and the University of Texas Postgraduate School of Medicine on March 6, 7, 8, 1960. Registration fee is $25.00 and the course is approved for 20 hours credit by AAPG. Further information is available by writing to Publications Office, Scott and White Clinic, Temple, Texas.

INSTITUTE IN PSYCHIATRY AND NEUROLOGY

February 25-26, 1960 **Little Rock, Arkansas**

The Twelfth Annual Institute in Psychiatry and Neurology will be held at the Consolidated VA Hospital, North Little Rock Division, Little Rock, Arkansas, on February 25-26, 1960. Additional information may be obtained by writing H. B. Witten, M.D., Assistant Director of Professional Services for Educaion, North Little Rock Division, Little Rock, Arkansas.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

March 7-10, 1960 **Roosevelt Hotel**

New Orleans, Louisiana

The twenty-third annual meeting of The New Orleans Graduate Medical Assembly will be held March 7-10, 1960 in New Orleans. Complete details may be obtained by writing to Mannie D. Paine, Jr., M.D., Secretary, The New Orleans Graduate Medical Assembly, 1430 Tulane Avenue, New Orleans 12, Louisiana.

Auxiliary News

25th Annual Doctors' Day

Definite planning has begun in county auxiliaries throughout southern states for our 25th annual observance of Doctors' Day. In fact, our *Sooner Physician's Wife*, through its excellent coverage of Doctors' Day, has helped stimulate interest in many states not included in the Southern Medical Association.

March 30 is the anniversary of the pioneer use of ether by Doctor Crawford W. Long—1842 was the year; Jefferson, Georgia, the place. In 1935 the first observation was held by a medical auxiliary. (Why not refresh your memory and re-read the interesting account in Fülöp-Miller's *Triumph Over Pain?* Do you remember the ether parties that led to Doctor Long's conclusions, eventual ostracism, and the dramatic conclusion?)

The variety of ways in which we celebrate March 30 is endless: with proclamations, red carnations, recognition in church services, and so on. The entertainments planned vary from formal Carnation Balls, with proceeds going to memorials, to chuck-wagon dinners. Some celebrate with solemnity, some with frivolity; regardless of manner, it is an occasion we all anticipate.

Our State Doctors' Day Chairman is Mrs. Lawrence E. Thompson, 4041 South Richmond, Tulsa.—And lest you forget, Mrs. Pat Fite, Sr., 1417 Emporia, Muskogee, is our State A.M.E.F. Chairman. What more timely way is there to thank our own family doctors than to make a contribution to A.M.E.F. in their honor?

Futures, Inc.

It is also timely, both in personal and organizational planning, to give much thought to strengthening our Paramedical Careers program. One auxiliary is planning a tea for the officers and sponsors of Future Nurses' Clubs in order to give them an opportunity to exchange ideas and evaluate their programs. Mrs. H. C. Manning, 1025 East Broadway, Cushing, is our Paramedical Recruitment Chairman, and has information available to answer any of your questions.

Meetings held before State Future Nurses' Day, March 19, should be geared to produce interest in attendance at the state meeting; those following this date should be of evaluation nature.

Auxiliaries should especially stress the availability of our Nurse Loan Fund program to every high school in the state, regardless of contact through organized clubs. Upon acceptance of any girl at any qualified school of nursing, application to our Nurse Loan Fund can be made immediately. Familiarity with the availability of this Fund may be the decisive factor in the plans of many spring graduates; and it cannot be over-emphasized. Mrs. William R. Cheatwood, Box 870, Duncan, is State Loan Fund Chairman.

AMERICAN COLLEGE OF ALLERGISTS

Graduate Instructional Course and Annual Congress

February 28-March 4, 1960 **The Americana Hotel**

Bal Harbor

Miami Beach, Florida

The American College of Allergists will hold a Graduate Instructional Course and Annual Congress February 28 to March 4, 1960 at the Americana Hotel, Bal Harbor, Miami Beach, Florida. For further information, contact John D. Gillaspie, M.D., Treasurer, 2049 Broadway, Boulder, Colorado.

PAN-PACIFIC SURGICAL ASSOCIATION

EIGHTH CONGRESS

September 27-October 5, 1960 **Honolulu, Hawaii**

The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, September 27 through October 5, 1960. All members of the profession are eligible to register.

Further information and brochures may be obtained by writing to F. J. Pinkerton, M.D., Director General of the Pan-Pacific Surgical Association, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.

PHYSICIAN PLACEMENT

Dermatology

Thomas E. Slimp, M.D., 323 West Livingston Place, Metairie, Louisiana, age 42, married, Syracuse Medical School, 1950, board certified January 1, 1960. No military obligations, available January 1, 1960.

Gordon Harold Ekblad, M.D., Field Medical Service School, Camp Pendleton, California, 53, married, University of Minnesota, 1930, available January 1960.

General Practice

Johnny Bill Delashaw, U.S.P.H.S. Hospital, Wyman Park Drive and 31st Street, Baltimore 11, Maryland, age 27, married, University of Texas Medical Branch, 1959, available July 1960.

D. H. Bessesen, M.D., Grand Canyon Hospital, Grand Canyon, Arizona, age 61, married, University of Minnesota, interested University Health Service, available October 1, 1959.

Sherman Allen Hope, M.D., 1105 Yale Ave., Panama City, Florida, PO 3-6656, age 27, married, University of Oklahoma School of Medicine, 1957, available July 1960.

Paul J. Vega, M.D., 6070 Orleans Ave., New Orleans, Louisiana, age 26, married, Louisiana State University, 1956, no military obligations, available now.

Thomas S. Whitecloud, M.D., 858 Market Street, Pascagoula, Mississippi, age 45, married, Tulane, 1943, veteran, has interest in teaching, availability date depends on situation.

Internal Medicine

Richard I. Hochman, M.D., 37, Badger Road, Annapolis, Maryland, 31, married, New York University, 1952, available October 1, 1960.

Albert D. Roberts, Jr., M.D., 4333 Colgate, Dallas, Texas, 29, married, Southwestern Medical School, Veteran, available July 1959.

Van B. Saye, Jr., M.D., 1230 Brockenbraugh Court, Metairie, Louisiana, age 28, married, Medical College of Georgia, 1954, Air Force Reserve, available July 1, 1960.

Locum Tenens

Harold W. Calhoon, M.D., Wesley Hospital, Oklahoma City. One year residency in Internal Medicine. Available July 1 to September 15th, 1960.

Arnold Giesbrecht, M.D., Hallock, Minnesota, age 34, married, graduate Winnipeg, Manitoba, 1957, prefers suburban or locality close to cities.

James D. Green, M.D., second year resident in Internal Medicine at St. John's Hospital, Tulsa, desires two weeks' tenure between now and July 1. Write to St. John's or call RIverside 3-3059.

Russell M. Preston, M.D., 476 San Juan, Oak Harbor, Washington, desires six month's work in General Practice between time of separation from military service to beginning of residency training, January-July, 1960.

Obstetrics and Gynecology

Lynn W. Abshere, M.D., 708 N.E. 14th, Oklahoma City, age 31, married, University of Oklahoma School of Medicine, 1956, American College of OB-GYN, no military obligations, available July 1, 1960.

Gerald R. Keilson, M.D., Medical Arts Building, Dallas, Texas, age 31, married, University of Texas, 1953, board qualified, veteran, available in July 1960.

Milton Gardner Mutch, Jr., M.D., 423 Mills Avenue, Fargo, North Dakota, age 31, married, University of Minnesota, 1955, board eligible upon completion of fellowship, veteran, available July 1, 1959.

David H. Holmes, M.D., 1017 Grovena Drive, St. Louis, Missouri, 37, married, Washington University, St. Louis, Veteran, available July 1, 1960.

Nejdat Mulla, M.D., 1110 Belmont Avenue, Youngstown, Ohio, age 36, married, University of Geneva, Switzerland, 1952, American College of OB-GYN, not eligible military service, available July 1, 1960.

Harold G. Ray, M.D., 212 Darien Place, Box 503, Balboa Heights, Canal Zone, age 28, married, University Arkansas School of Medicine, 1955, now in active reserve, available July 1, 1960.

Alvon C. Winegar, M.D., 1940 Michigan, Benton Harbor, Michigan, age 41, married, Wayne University, 1942, no military obligation, available early 1960.

Deaths

Harley M. Prentiss, M.D.
1892-1959

Harley M. Prentiss, M.D., 67-year-old Nowata physician, died in Rochester, Minnesota on December 26, 1959.

A native of Metamora, Ohio, Doctor Prentiss graduated from the University of Oklahoma School of Medicine in 1918. He established his practice in Yale, Oklahoma in 1920, later moving to Nowata and then to Roosevelt, Oklahoma. He returned to Nowata in 1948.

Doctor Prentiss was a Past-Secretary of the Washington-Nowata County Medical Society.

Jack W. Myers, M.D.
1917-1960

Jack W. Myers, M.D., 43-year-old El Reno physician, died at his home on January 3, 1960.

Born in Apache, Oklahoma, Doctor Myers graduated from the University of Oklahoma School of Medicine in 1943. After taking a residency at the City-County Hospital in Fort Worth, Texas, he established his practice in El Reno.

Doctor Myers served in the army medical corps during World War II and with the air force in the Korean conflict.

John Reynolds, M.D.
1874-1959

John Reynolds, pioneer Muskogee physician, died December 28, 1959 in Muskogee.

A native of Asheville, North Carolina, Doctor Reynolds graduated from the University of Louisville Medical College in 1903. All but one year of his 57-year medical career was spent in Muskogee.

In 1952, Doctor Reynolds was honored for his years of service to the medical profession when the Oklahoma State Medical Association presented him a 50-year pin.

William A. Walker, M.D.
1892-1959

William A. Walker, M.D., 67-year-old Tulsa physician, died December 31, 1959 in Tulsa.

Born in Holdenville, Doctor Walker graduated from the University of Oklahoma School of Medicine in 1927. After establishing his practice in Tulsa, he served as public schools physician for eight years.

Doctor Walker was a member of the Oklahoma Chapter, American Academy of General Practice.

Pediatrics

H. M. McClintock, M.D., 5712 St. John St., Kansas City, Missouri, 29, married, Baylor University, 1955, veteran, available December 1960.

Surgery

(Name on Request) 32 years old, married, Tulane, 1952, veteran, board eligible.

Olin O. Williams, Jr., M.D., 3768 Alma Drive, Memphis, Tennessee, 29, married, University of Tennessee, veteran, available January 1962.

Urology

Charles Lee Reynolds, Jr., M.D., 5012 Rockport Drive, Dallas, Texas, age 32, Southwestern Medical College, Dallas, 1949, board eligible, married, no military obligations, available July 15, 1960.

Alex Grossman, M.D., V.A. Center, Temple, Texas, 42, married, University of Texas, 1951, board certified, veteran, available now.

Herbert Sohn, M.D., 2010 Cliffview Road, Cleveland, Ohio, 32, married, Chicago Medical School, 1955, veteran, available July 1960.

MISCELLANEOUS ADVERTISEMENTS

FOR SALE: Autoclave, Castle, electric. Originally purchased for $1866.75 in 1956. Contact Walt Allen, 116 North Fourth Street, Chicaksha, Oklahoma.

FOR SALE or RENT: Office building, equipment and supplies, including x-ray. Residence also available. Contact Box F, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: General practice established 7 years in prosperous oil and agriculture community. Town is 5000 and trade area over 15,000 population. Gross receipts over $50,000 annually. New clinical building for sale or rent. New community hospital expected within a year. $30,000 for practice, equipment and current accounts receivable. Terms. Owner going into residency. Contact Box L, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: $40,000 per year General Practice. 14 room completely equipped clinic. County seat town of 4,000 with new 40 bed hospital. Liberal terms. Write Key J, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

OFFICE SPACE for rent, modern building, 528 N.W. 12th, Oklahoma City. Complete maintenance, air conditioning, free parking space for patients. CE 6-1631.

WANTED: Internist to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR RENT: Office space for two doctors, modern, air conditioned building. 3832 N. May Ave., Oklahoma City. Complete maintenance. Free parking space for patients. WI 2-5531.

WANTED: Surgeon to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

WILL CUSTOM BUILD offices for physicians and dentists in new medical center located on North Western in Oklahoma City. Approximately 500 square feet per unit. Ample parking. Construction to start immediately. Contact Bob Fowler, P.O. Box 9554, Shartel Station, Oklahoma City, WI 2-1021.

WANTED: General Practitioner with some ability to do surgery. Primarily industrial practice. Beginning salary $900.00. Contact C. J. Paternostro, M.D., 426 Medical Arts Building, Dallas, Texas.



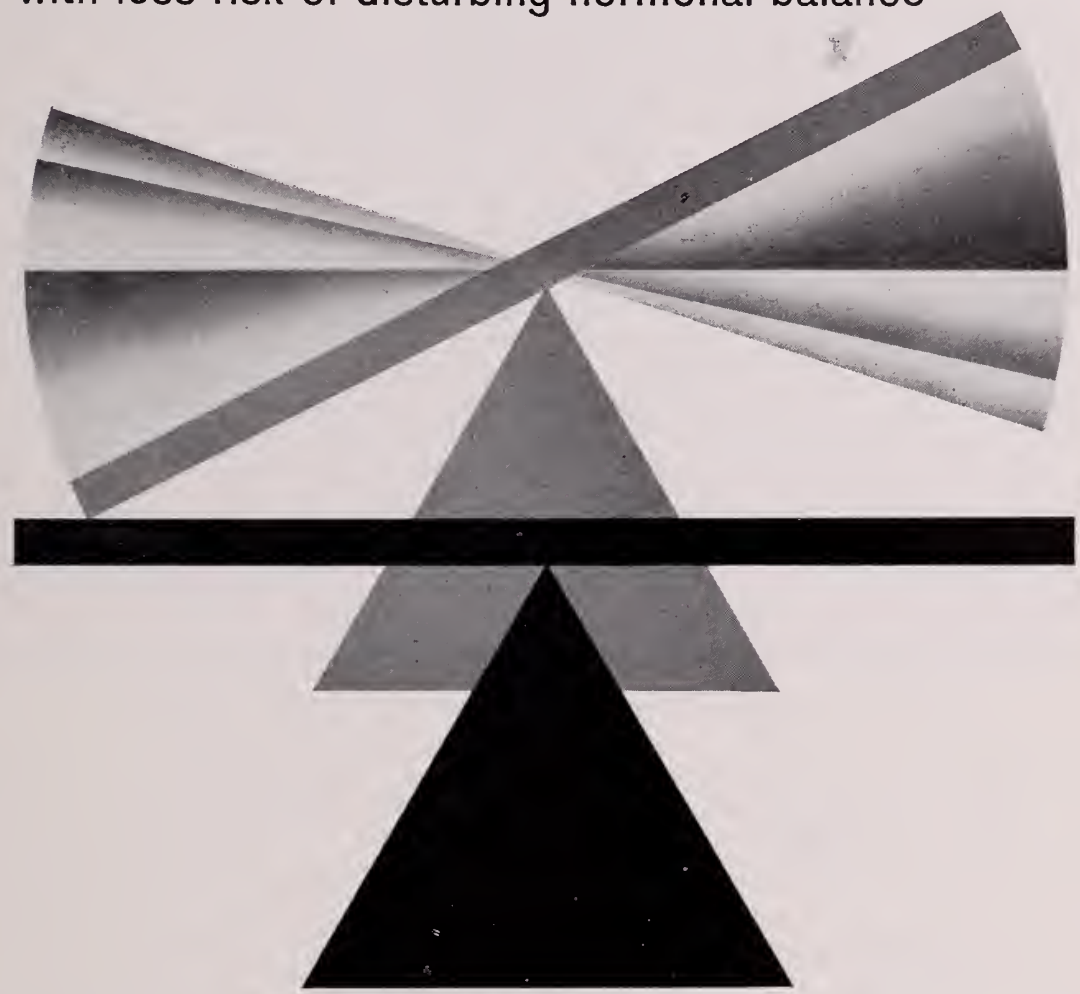
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In the treatment of the rheumatic disorders new Sterazolidin provides a method of limiting the gravest danger inherent in steroid therapy... hypercortisonism arising from excessive dosage.

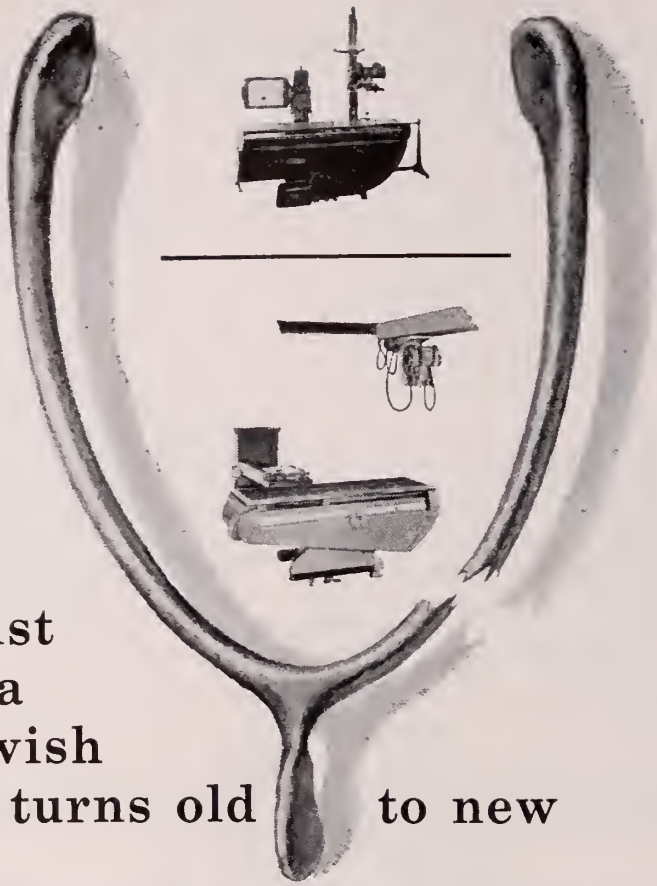
Repeatedly it has been shown that the addition of low dosage of Butazolidin sharply reduces hormone requirement.[1-4] Sterazolidin is a combination of prednisone (1.25 mg.) and Butazolidin (50 mg.) which provides, in the majority of cases, consistent relief at a stable uniform maintenance dosage significantly below the level at which serious hormonal imbalance is likely to occur.

Sterazolidin® (prednisone-phenylbutazone Geigy). Each capsule contains prednisone 1.25 mg.; phenylbutazone 50 mg.; dried aluminum hydroxide gel 100 mg.; magnesium trisilicate 150 mg. and homatropine methylbromide 1.25 mg.

1. Kuzell, W. C., and others.: Arch. Int. Med. 92:646, 1953. 2. Wolfson, W. Q.: J. Michigan M. Soc. 54:323, 1955. 3. Strandberg, B.: Brit. J. Phys. Med. 19:9, 1956. 4. Platt, W. D., Jr., and Steinberg, I. H.: New England J. Med. 256:823 (May 2) 1957.

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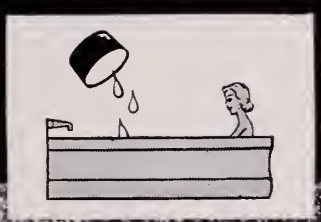
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Patients will appreciate pleasant, convenient, easy to use, pine-scented SARDO. Non-sensitizing. Most economical. Bottles of 4, 8 and 16 oz.

1. Spoor, H. J.: N. Y. State J. Med. Oct. 15, 1958

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in the bath

for atopic dermatitis
eczematoid dermatitis
senile pruritus
contact dermatitis
soap dermatitis



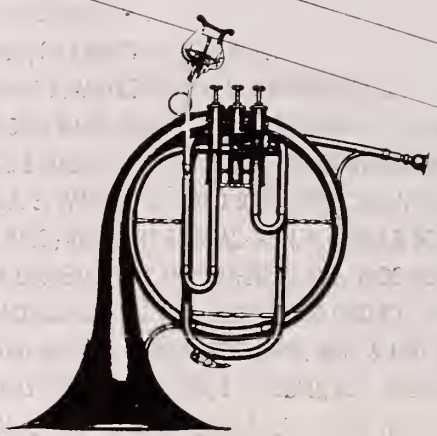
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Announcing an Old Product

THAT STILL IS THE MOST IMPROVED AGENT FOR BASIC DRUG THERAPY IN HYPERTENSION



Only time and clinical acceptance truly define whether a drug is safe and effective. Of the many Rauwolfia compounds, there is one alkaloidal fraction capable of producing antihypertensive benefits with minimal side effects, purified alseroxylon complex (RAUTENSIN®).[1-6] This compound is less likely to cause such side effects as mental depression, lethargy, listlessness, and drowsiness consistently reported with reserpine.

IN MILD HYPERTENSION

RAUTENSIN®

(Tablets containing 2 mg. purified alseroxylon complex)

IN MODERATE TO SEVERE HYPERTENSION

RAUVERA®

(Tablets containing 1 mg. Rautensin and 3 mg. alkavervir)

Rautensin bibliography: 1. WRIGHT, W. T., JR.; POKORY, C., AND FOSTER, T.: AM. PRACT. & DIGEST. TREAT. 7:1992, 1956. 2. SUCKLE, E.: GERIATRICS 11:509, 1956. 3. FINCH, W. J.: J. OKLAHOMA M.A. 50:259, 1957. 4. TERMAN, L. A.: ILLINOIS M. J. 3:67, 1957. 5. GIFFORD, R. W.: J. ARKANSAS M. SOC. 55:31, 1958. 6. FORD, R. V., AND MOYER, J. H.: POSTGRAD. MED. 23:41, 1958.

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CUT DOWN ON
that 1½ Billion Days of Illness Due to
COLDS—

NEO-SYNEPHRINE® COMPOUND
COLD TABLETS

for "Syndromatic" Control of the Common Cold and Allergic Rhinitis

provide **PROTECTION**— *through the full range of cold symptoms*

PROTECTION from Nasal Stuffiness
— Neo-Synephrine HCl, 5 mg. — first choice in decongestants.

PROTECTION from Aches, Fever
— Acetaminophen, 150 mg. — modern analgesic, antipyretic.

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— Thenfadil® HCl, 7.5 mg. — effective antihistaminic.

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— Caffeine, 15 mg. — dependable, mild stimulant.

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DOSAGE: Adults—2 tablets three times daily.
Children from 6 to 12 years—
1 tablet three times daily.

Bottles of 20 and 100 tablets.

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COMPOCILLIN®-VK
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OFFERS THE HIGH BLOOD LEVELS OF POTASSIUM PENICILLIN V

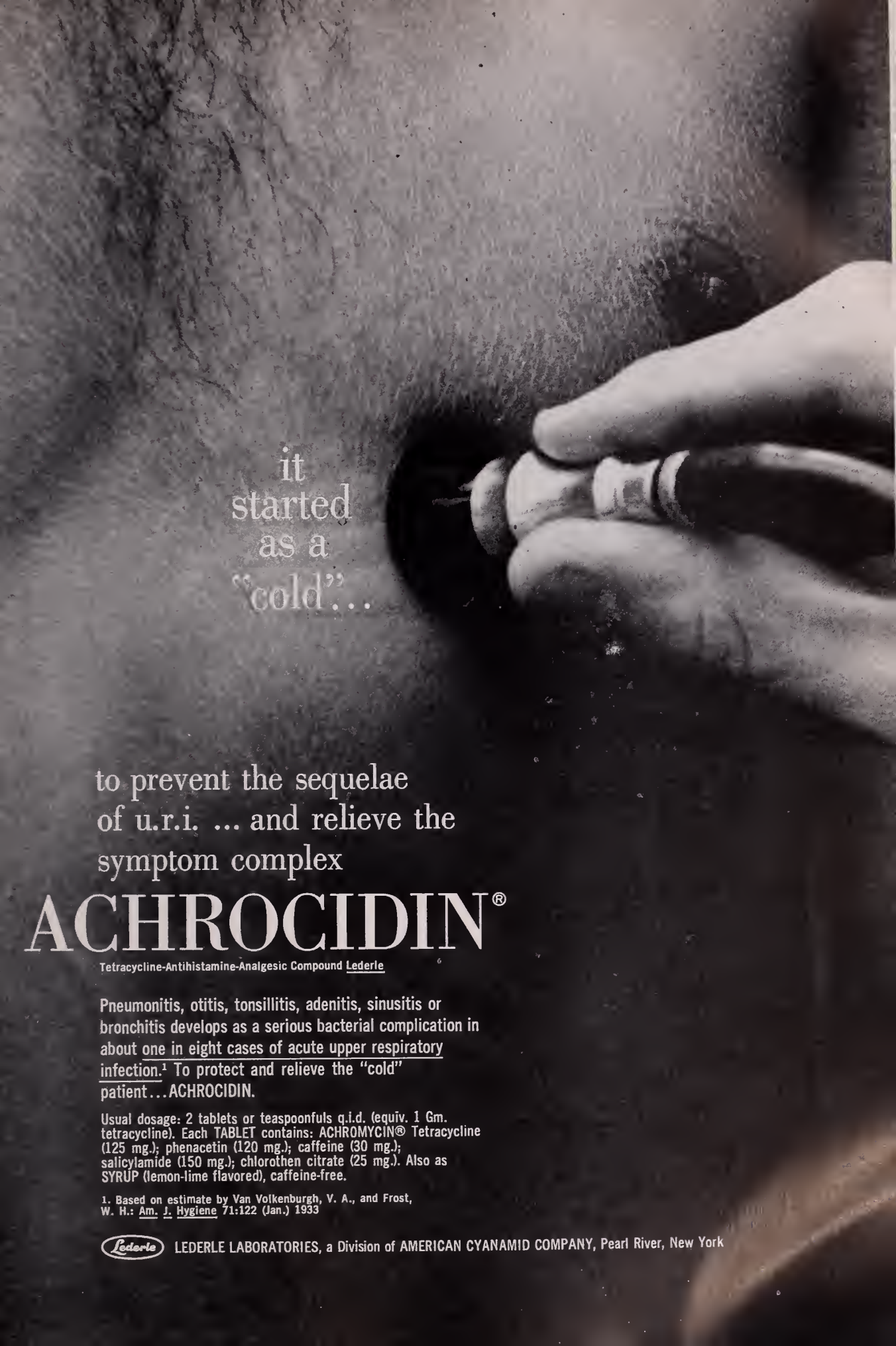


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to prevent the sequelae
of u.r.i. ... and relieve the
symptom complex

ACHROCIDIN®

Tetracycline-Antihistamine-Analgesic Compound Lederle

Pneumonitis, otitis, tonsillitis, adenitis, sinusitis or bronchitis develops as a serious bacterial complication in about one in eight cases of acute upper respiratory infection.[1] To protect and relieve the "cold" patient...ACHROCIDIN.

Usual dosage: 2 tablets or teaspoonfuls q.i.d. (equiv. 1 Gm. tetracycline). Each TABLET contains: ACHROMYCIN® Tetracycline (125 mg.); phenacetin (120 mg.); caffeine (30 mg.); salicylamide (150 mg.); chlorothen citrate (25 mg.). Also as SYRUP (lemon-lime flavored), caffeine-free.

1. Based on estimate by Van Volkenburgh, V. A., and Frost, W. H.: Am. J. Hygiene 71:122 (Jan.) 1933

Lederle LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York



NEOCHOLAN®

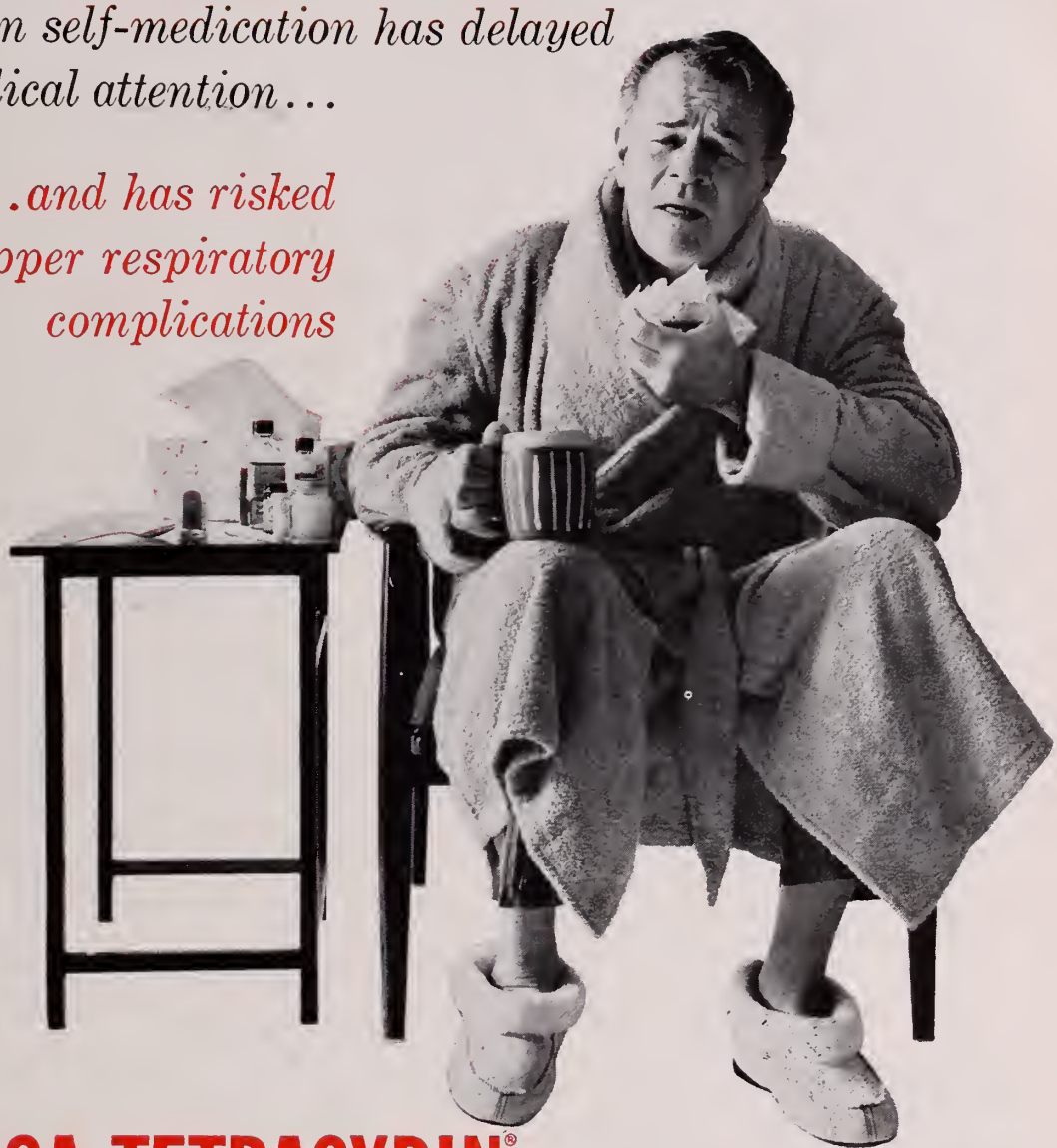
Your patient will often respond promptly to Neocholan therapy. It greatly increases the flow of thin, nonviscid bile and corrects biliary stasis by flushing the biliary system. It also relaxes intestinal spasm, resulting in an unimpeded flow of bile and pancreatic juice into the small intestine. Neocholan helps to promote proper digestion and absorption of nutrients. It also encourages normal peristalsis by restoring intestinal tone.

Each Neocholan tablet provides:
Dehydrochloric Acid Compound, P-M Co. 265 mg. (Dehydrochloric Acid, 250 mg.); Homatropine methylbromide 1.2 mg.;Phenobarbital 8.0 mg.
Supplied in bottles of 100 tablets.

PITMAN-MOORE COMPANY
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in the **"COMMON COLD"**
when self-medication has delayed medical attention...

...and has risked upper respiratory complications



COSA-TETRACYDIN® CAPSULES

Cosa-Tetracyn® — analgesic — antihistamine compound

act quickly to

- *control secondary infection*
- *alleviate cold symptoms*

each capsule contains:

Cosa-Tetracyn	125 mg.
phenacetin	120 mg.
caffeine	30 mg.
salicylamide	150 mg.
buclizine HCl	15 mg.

average adult dose: 2 capsules q. i. d.

Pfizer *Science for the world's well-being*

PFIZER LABORATORIES, *Division, Chas. Pfizer & Co., Inc., Brooklyn 6, N.Y.*

reaches all nasal and paranasal membranes *systemically*[1]



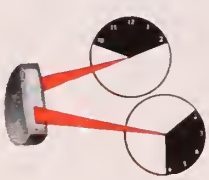
Pharmacologically balanced formula for prompt symptomatic relief

- in nasal and paranasal congestion
- in sinusitis and postnasal drip
- in allergic reactions of the upper respiratory tract

Triaminic*[2,3] *is safer and more effective than topical medication

- transported systemically to *all* respiratory membranes
- provides longer-lasting relief
- presents no problem of rebound congestion
- avoids "nose drop addiction"

Relief is prompt and prolonged because of this special timed-release action:



first — the outer layer dissolves within minutes to produce 3 to 4 hours of relief

then — the core disintegrates to give 3 to 4 *more* hours of relief

Each Triaminic timed-release Tablet provides:
Phenylpropanolamine HCl.....50 mg.
Pheniramine maleate.....25 mg.
Pyrilamine maleate.....25 mg.

Dosage: 1 tablet in the morning, midafternoon and at bedtime. In postnasal drip, 1 tablet at bedtime is usually sufficient.

Each timed-release Triaminic Juvelet® provides: ½ the formulation of the Triaminic Tablet.

Dosage: 1 Juvelet in the morning, midafternoon and at bedtime.

Each tsp. (5 ml.) of Triaminic Syrup provides: ¼ the formulation of the Triaminic Tablet.

Dosage (to be administered every 3 or 4 hours): *Adults* — 1 or 2 tsp.; *Children 6 to 12* — 1 tsp.; *Children 1 to 6* — ½ tsp.; *Children under 1* — ¼ tsp.

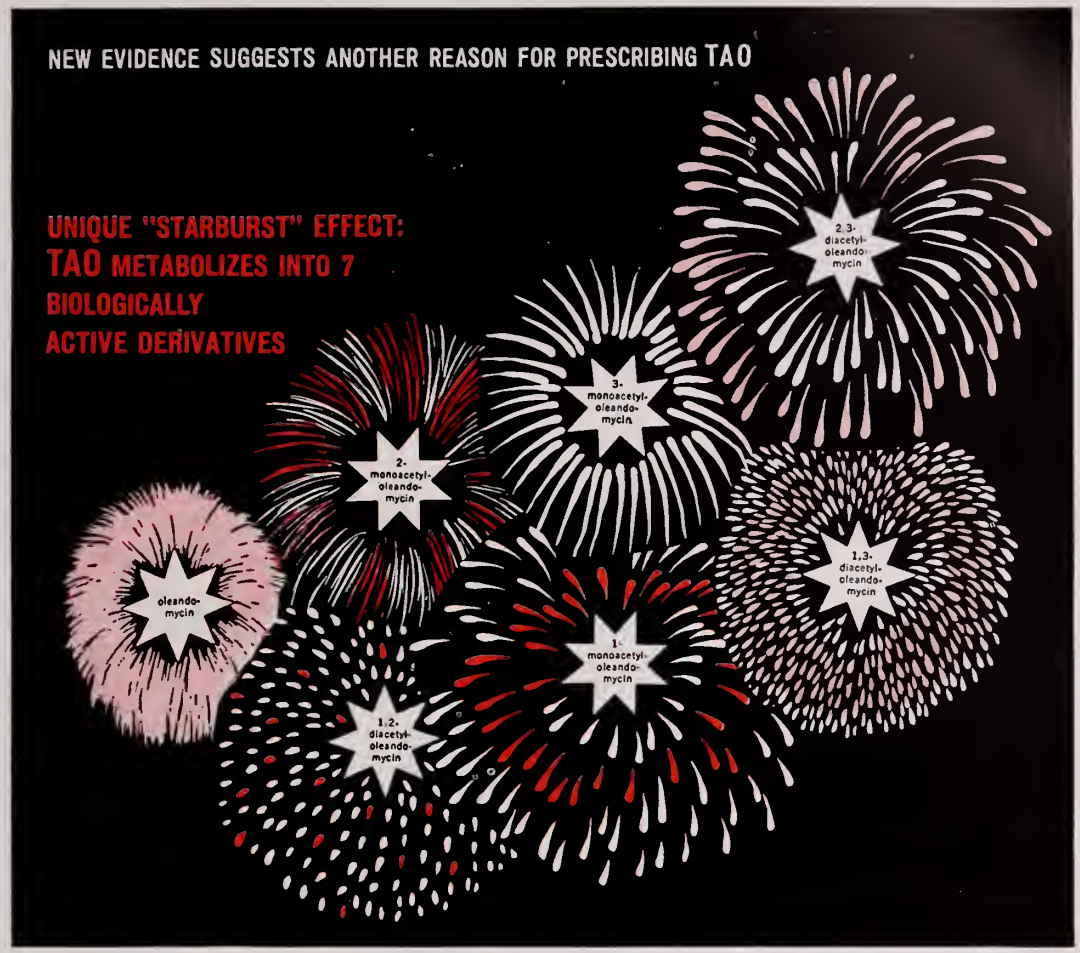
1. Fabricant, N. D.: E.E.N.T. Monthly *37*:460 (July) 1958.
2. Lhotka, F. M.: Illinois M. J.: *112*:259 (Dec.) 1957.
3. Farmer, D. F.: Clin. Med. *5*:1183 (Sept.) 1958.

the leading oral nasal decongestant...

Triaminic®

timed-release tablets and juvelets
also non-alcoholic, fruit-flavored syrup

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The impression that TAO is an unusually active antibiotic has steadily gained recognition by impressive clinical performance. Now come reports of **in vivo** and **in vitro** biological and biochemical evaluations that show TAO to be indeed unique.[1,2]

TAO differs from other antibiotics in that it is metabolized to multiple active compounds which **remain** active throughout their presence in the body. These 7 derivatives (in addition to TAO) show activity against common Gram-positive pathogens, including resistant strains of **Staph. aureus.**

In light of these findings, take another look at TAO performance: • 92% success in published cases of Gram-positive respiratory, skin, soft tissue and genitourinary infection • Effective against 78% of 64 "antibiotic-resistant" epidemic staphylococci. (In the same study, chloramphenicol was active against 52%; erythromycin against only 25%)[3] • No side effects in 94%; infrequent reactions mild and easily reversed • Quickly absorbed • Highly palatable.

Sound reasons to: Start with TAO to end 9 out of 10 common Gram-positive infections.

Supplied: TAO Capsules—250 mg., and 125 mg., bottles of 60. **TAO for Oral Suspension**—125 mg. per tsp. (5 cc.) When reconstituted; unusually palatable cherry flavor; 60 cc. bottle. Prescription only.

Other TAO forms available: TAO Pediatric Drops: flavorful, easy to administer. **TAO®-AC:** TAO analgesic, antihistaminic compound. **TAOMID®:** TAO with triple sulfas. **Intramuscular or Intravenous:** in clinical emergencies. Prescription only.

1. English, A. R., and McBride, T. J.: Proc. Soc. Exper. Biol. & Med. 100:880 (Apr.) 1959. 2. Celmer, W. D.: Antibiotics Annual 1958-1959, New York, Medical Encyclopedia, Inc., 1959, p. 277. 3. English, A. R., and Fink, F. C.: Antibiotics & Chemother. 8:420 (Aug.) 1958.

designed for superior control of common Gram-positive infections

TAO®

(triacetyloleandomycin)
Capsules/Oral Suspension



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Division, Chas. Pfizer & Co., Inc.
Science for the World's Well-Being



whenever there is inflammation, swelling, pain

VARIDASE®

STREPTOKINASE-STREPTODORNASE LEDERLE

BUCCAL *Tablets*

conditions for a fast comeback . . .



5 days of classic therapy — after 48 hours of VARIDASE

as in cellulitis*

Until VARIDASE stemmed infection, inflammation, swelling and pain, neither medication nor incision and drainage had affected the increasing cellulitis.

VARIDASE mobilizes the natural healing process, by accelerating fibrinolysis, to condition the patient for successful primary therapy. Increases the penetrability of the fibrin wall, for easy access by antibodies and drugs . . . without destroying limiting membrane . . . and limits infiltration. Prescribe VARIDASE Buccal Tablets routinely in infection or injury.

*Innerfield, I.: Clinical report cited with permission.

VARIDASE BUCCAL Tablets contain:
10,000 Units Streptokinase, 2,500 Units Streptodornase.

Supplied: Boxes of 24 and 100 tablets

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in every arthritic state
long-term therapy
with maximum relief of pain
without steroids

Buffered

Pabirin®

Tablets

improved salicylate therapy

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska

This is Panalba performance...





in sinusitis

. . . into a mixed culture of the four organisms commonly involved in sinusitis . . . *Str. hemolyticus, D. pneumoniae, H. influenzae* and *Staph. aureus* (in this case a resistant strain) . . . we introduce the five most frequently used antibiotics.

Twenty-four hours later (in this greatly enlarged photograph), note that only *one* of the five leading antibiotics has stopped *all* the organisms, including the resistant staph! This is Panalba.

In your next patient with sinusitis . . . in *all* your patients with potentially-serious infections . . . provide this extra protection with your prescription:

Dosage—1 or 2 capsules 3 or 4 times a day.
Supplied—Capsules containing Panmycin phosphate equivalent to 250 mg. tetracycline hydrochloride, and 125 mg. Albamycin as novobiocin sodium, in bottles of 16 and 100.
Now available: new Panalba Half-Strength Capsules in bottles of 16 and 100.

Panalba*

(Panmycin* Phosphate plus Albamycin*)

The broad-spectrum antibiotic of *first* resort



Upjohn The Upjohn Company Kalamazoo, Michigan

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no irritating crystals[1] · uniform concentration in each drop[2]

STERILE OPHTHALMIC SOLUTION

NEO-HYDELTRASOL®

PREDNISOLONE 21-PHOSPHATE-NEOMYCIN SULFATE

2,000 TIMES MORE SOLUBLE THAN PREDNISOLONE OR HYDROCORTISONE

"The solution of prednisolone has the advantage over the suspension in that no crystalline residue is left in the patient's cul-de-sac or in his lashes . . . . The other advantage is that the patient does not have to shake the drops and is therefore sure of receiving a consistent dosage in each drop."[2]

1. Lippmann, O.: Arch. Ophth. **57**:339, March 1957.
2. Gordon, D.M.: Am. J. Ophth. **46**:740, November 1958.

supplied: 0.5% Sterile Ophthalmic Solution NEO-HYDELTRASOL (with neomycin sulfate) and 0.5% Sterile Ophthalmic Solution HYDELTRASOL. In 5 cc. and 2.5 cc. dropper vials. Also available as 0.25% Ophthalmic Ointment NEO-HYDELTRASOL (with neomycin sulfate) and 0.25% Ophthalmic Ointment HYDELTRASOL. In 3.5 Gm. tubes.

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Lysine-Vitamins Lederle

help restore the normal blood picture—iron as ferric pyrophosphate to restore or maintain normal hemoglobin.

boost appetite and energy—vitamins . . . B_1, B_6 and B_{12}.

upgrade low-grade protein—cereals and other low protein favorites of children, upgraded by l-Lysine, work with meat and other top protein to build stronger bodies.

tastes good! Each daily cherry-flavored teaspoonful dose (5 cc.) contains:

l-Lysine HCl	300 mg.
Vitamin B_{12} Crystalline	25 mcgm.
Thiamine HCl (B_1)	10 mg.
Pyridoxine HCl (B_6)	5 mg.
Ferric Pyrophosphate (Soluble)	250 mg.
Iron (as Ferric Pyrophosphate)	30 mg.
Sorbitol	3.5 Gm.
Alcohol	0.75%

Bottles of 4 and 16 fl. oz.

Lederle LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

DOES YOUR PRESENT ANTICHOLINERGIC REALLY



The test—you might say the acid test—of an anticholinergic is simple: will it protect your patient from hyperacidity around the clock, **even while he sleeps.** The weakness of t.i.d. or q.i.d. preparations is well recognized; but even some "b.i.d." encapsulations may be unreliable. McHardy, for instance, found a "widely variable duration of action, definitely less than that anticipated" in the "sustained," "delayed," and "gradual release" anticholinergics he studied.[1]

COMPARE THE DATA ON ENARAX . . . the new combination of an **inherently** long-acting anticholinergic (oxyphencyclimine) and Atarax, the non-secretory tranquilizer. Note the effectiveness of oxyphencyclimine:

OBSERVE THE OXYPHENCYCLIMINE REPORTS . . .

McHardy: "[Oxyphencyclimine] has proved to be an excellent sustained-action anticholinergic in our study of this agent over a period of eighteen months."[1]

Kemp: ". . . for the majority of patients, one tablet every 12 hours provided adequate control. This characteristic long action . . . may constitute an advantage of this drug as compared to coated 'long-acting' preparations of other compounds."[3]

Add Atarax to this 12-hour anticholinergic. The resulting combination—ENARAX—now gives relief from emotional stress, in addition to a reduction of spasm and acid. Atarax does not stimulate gastric secretion. No serious adverse clinical reaction has ever been documented with Atarax.

LOOK AT THE RESULTS WITH ENARAX[4,5]:

Does the medication you now prescribe assure you of all these benefits? If not, why not put your next patient with peptic ulcer or G.I. dysfunction on therapy that **does.**

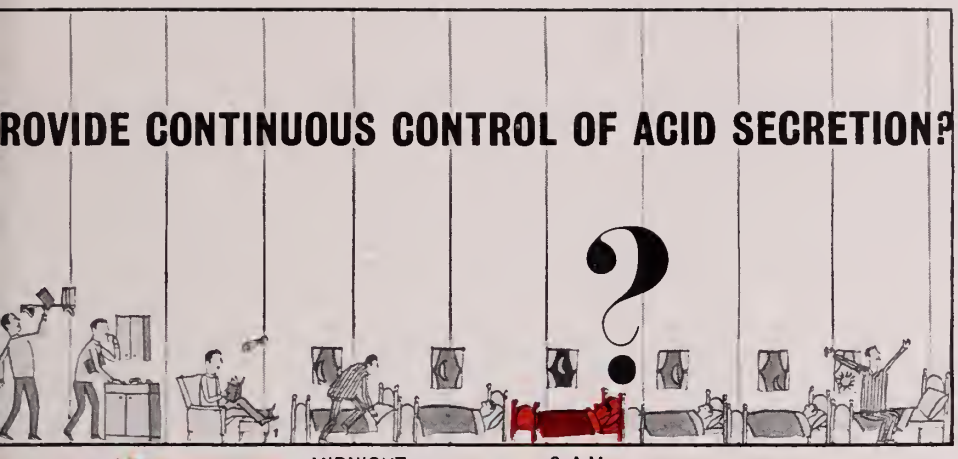
ENARAX®



(oxyphencyclimine plus ATARAX®)

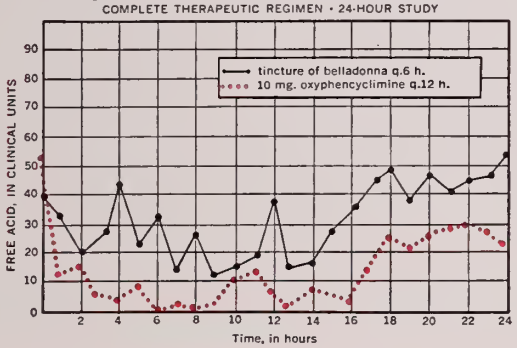
A SENTRY FOR THE G.I. TRACT

ROVIDE CONTINUOUS CONTROL OF ACID SECRETION?



"Prolonged periods of achlorhydria" after 10 mg. oxyphencyclimine q. 12 h.[3]

MEAN GRAPH OF GASTRIC ACIDITY IN 4 PATIENTS RECEIVING COMPLETE THERAPEUTIC REGIMEN • 24-HOUR STUDY



Clinical Diagnosis: Peptic Ulcer—Gastritis—Gastroenteritis—Colitis—Functional Bowel Syndrome—Duodenitis—Hiatus Hernia (symptomatic)—Irritable Bowel Syndrome—Pylorospasm—Cardiospasm—Biliary Tract Dysfunctions—and Dysmenorrhea.

Clinical Results: Effective in over 92% of cases.

As for Safety: "Side reactions were uncommon, usually no more than dryness of the mouth...."[4]

Each ENARAX tablet contains:

Oxyphencyclimine HCl ..... 10 mg.
Hydroxyzine (ATARAX®) ..... 25 mg.

Dosage: One-half to one tablet twice daily—preferably in the morning and before retiring. The maintenance dose should be adjusted according to therapeutic response. Use with caution in patients with prostatic hypertrophy and with ophthalmological supervision only in glaucoma.

Supplied: In bottles of 60 black-and-white scored tablets.

References: 1. McHardy, G., et al.: J. Louisiana M. Soc. 111:290 (Aug.) 1959. 2. Steigmann, F.: Study conducted at Cook County Hospital, Chicago, Illinois, in press. 3. Kemp, J. A.: Antibiotic Med. & Clin. Therapy 6:534 (Sept.) 1959. 4. Leming, B. H., Jr.: Clin. Med. 6:423 (Mar.) 1959. 5. Data in Roerig Medical Department files.

ROERIG Rx

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Science for the World's Well-Being™

The first synthetic penicillin
available
for general clinical use

FOR YOUR NEXT PATIENT WHERE PENICILLIN IS INDICATED

SYNC

BLOOD LEVELS TWICE AS HIGH AS WITH POTASSIUM PENICILLIN V

ORAL ROUTE PROVIDES HIGHER BLOOD LEVELS THAN INTRAMUSCULAR PENICILLIN G

IMPROVED ANTIBIOTIC ACTION FROM ISOMERIC COMPLEMENTARITY





CONSIDER THESE 6 IMPORTANT THERAPEUTIC BENEFITS OF

SYNCILLIN™



POTASSIUM PENICILLIN-152

ANTIBIOTIC ACTIVITY DIRECTLY PROPORTIONAL TO ORAL DOSE

REDUCED RATE OF INACTIVATION BY STAPH PENICILLINASE

MANY STAPH STRAINS MORE SENSITIVE TO SYNCILLIN IN VITRO

BRISTOL





FOR HIGHLY EFFECTIVE THERAPY OF THE LARGE VARIETY OF INFECTIONS CAUSED BY SUSCEPTIBLE PATHOGENS... NEW

SYNC

Significance of complementary action of isomers in SYNCILLIN

The antibiotic effect of the clinically available mixture, SYNCILLIN, is greater than that of either of its two component isomers alone against many important pathogens, including some penicillin-resistant staphylococci. This phenomenon has been described as *Isomeric Complementarity*.

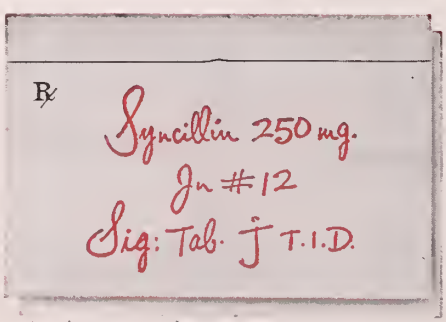
Significance of higher blood levels with SYNCILLIN

Higher blood levels may be of value with organisms of only moderate penicillin sensitivity where doubling the blood concentration may be essential for effective bactericidal action. In addition, these higher levels may be necessary where there is infection in areas with a poor blood supply.[6] Under these circumstances a higher blood concentration may provide the increased diffusion pressure required to deliver adequate amounts to the tissue. Also, antibiotic activity of SYNCILLIN is directly proportional to oral dosage. Increasing the dosage may, therefore, enhance the drug's effectiveness in certain cases.

Efficacy of SYNCILLIN against staphylococci and other resistant organisms

Studies have shown that SYNCILLIN is effective *in vitro* against 60 to 75% of hospital "staph" strains, while penicillin G and penicillin V are now effective against only 30 to 50%.[1, 2] Therefore, if clinical judgment indicates the use of penicillin, SYNCILLIN would be expected to be the most effective. However, since some strains are still resistant to SYNCILLIN as well as to the other penicillins, cultures and sensitivity tests should be performed where indicated by clinical judgment.

There have recently been reports of decreased efficacy of penicillin in streptococcal[3] and gonococcal[4, 5] infections. The emergence of penicillin-resistant gonococci appears to be associated with an increase in the incidence of gonorrhea all over the world. When a less sensitive strain is encountered the higher blood levels produced by SYNCILLIN may be most helpful.



major therapeutic advantages accompany molecular asymmetry

I L L I N™

Relation of intermittent high blood levels of SYNCILLIN to antibacterial efficacy

SYNCILLIN, like all clinically available penicillins, is bactericidal. Periodic high blood concentrations are sufficient to permit complete eradication of sensitive pathogens. Continuous high blood levels are not required with SYNCILLIN. According to Eagle,[7] "Soon after penicillin attains effective concentrations, the bacteria cease multiplying; and the bacteriostatic effect persists for a number of hours after penicillin has fallen to concentrations that are wholly ineffective....The therapeutic significance of this postpenicillin recovery period is enhanced by the fact that the recovering bacteria, damaged but not killed by the previous exposure to penicillin, are abnormally susceptible to the host defenses. In consequence, the bactericidal process *in vivo* continues for many hours after the drug itself has fallen to ineffective concentrations."

Reduced rate of inactivation of SYNCILLIN by staph penicillinase

Bacterial resistance to penicillin has been attributed to the action of penicillin-inactivating enzymes produced by the invading organisms. SYNCILLIN is less affected by staphylococcal penicillinase than either of its component isomers. Further, SYNCILLIN is shown to be less inactivated by this enzyme than penicillin V and penicillin G. Penicillinase from *B. cereus* likewise inactivates SYNCILLIN less rapidly than penicillin V and G. But this would not impede the therapeutic use of this penicillinase in allergic reactions. This is because the massive dosage with which this enzyme is administered would effectively destroy SYNCILLIN in the body.

References: 1. Wright, W. W.: Microbiology Report to Bristol Laboratories Inc. 2. Kligman, A.; Morigi, E. M. E.; Wheatley, W. B., and Albright, H.: Paper presented at the Seventh Antibiotic Symposium, November 4-6, Washington, D.C. 3. Editorial: New England J. Med. **261**:305 (Aug. 6) 1959. 4. King, A.: Lancet **1**:651 (March 29) 1958. 5. Epstein, E.: J.A.M.A. **169**:1055 (March 7) 1959. 6. Kass, E. H.: Am. J. Med. **18**:764 (May) 1955. 7. Eagle, H.: J. Bact. **58**:475, 1949.

Indications: SYNCILLIN is recommended in the treatment of infections caused by pneumococci, streptococci, gonococci, corynebacteria, and penicillin-sensitive staphylococci. In addition, SYNCILLIN is effective against certain strains of staphylococci resistant to other penicillins. SYNCILLIN, like other oral penicillins, is not recommended at the present time in deep-seated or chronic infections, subacute bacterial endocarditis, meningitis, or syphilis.

Dosage: 125 mg. or 250 mg. three times daily, depending on the severity of infection. Larger doses (e.g., 500 mg. t.i.d.) may be used for more severe infections. SYNCILLIN may be administered without regard to meals. Beta hemolytic streptococcal infections should be treated with SYNCILLIN for at least ten days.

Precautions: At the present time it is not possible to draw definite conclusions regarding the incidence of allergenicity to SYNCILLIN or its cross-allergenicity with natural penicillins. *Therefore, the usual precautions for oral penicillin therapy should always be observed.* Patients with histories of asthma, hay fever, urticaria, or previous reactions to penicillin should be watched with special care. Administration of oral penicillin, in rare instances, may provoke acute anaphylaxis, particularly in penicillin-sensitive individuals.
Diarrhea has been reported occasionally following heavy dosage. If this occurs, lengthen the interval between dosages.
If superinfection occurs during therapy, appropriate measures should be taken. Since some strains of staphylococci are resistant to SYNCILLIN as well as to other penicillins, cultures and sensitivity tests should be performed where indicated by clinical judgment. As is true with all antibiotics, clinical response does not always correlate with laboratory bacterial sensitivity reports.

Supply: 125 and 250 mg. tablets, bottles of 25 and 100. 125 mg. powder for oral solution, 60 ml. vials.

BRISTOL

BRISTOL LABORATORIES, Division of Bristol-Myers Company, SYRACUSE, NEW YORK

NIAMID
the mood brightener

makes the cancer patient more comfortable

- reduces impact of pain
- decreases narcotic requirements
- increases appetite
- improves mental outlook

NIAMID lessens the need for narcotics in the depressed cancer patient and appears to potentiate pain-relieving agents. As pain is reduced and mental outlook improves, apprehension and depression are replaced by a brighter and more alert attitude, and appetite returns. The family, too, is cheered by the improvement in the patient's condition. With NIAMID therapy, patient care becomes noticeably less demanding.

Supply: NIAMID (brand of nialamide) is available as 25 mg. (pink) and 100 mg. (orange) scored tablets.

Complete references and a Professional Information Booklet giving detailed information on NIAMID are available on request from the Medical Department, Pfizer Laboratories, Division, Chas. Pfizer & Co., Inc., Brooklyn 6, New York

NIAMID®
the mood brightener in cancer

Pfizer

Science for the world's well-being™





Diagnostic Quandaries

Colitis? Gall Bladder Disease?
Chronic Appendicitis?
Rheumatoid Arthritis? Regional Enteritis?

A DISEASE that is frequently overlooked in solving diagnostic quandaries is amebiasis. Its symptoms are varied and contradictory, and diagnosis is extremely difficult. In one study, 56% of the cases would have been overlooked if the routine three stool specimens had been relied on.[1]

Another study found 96% of a group of 150 patients with rheumatoid arthritis were infected by *E. histolytica*. In 15 of these subjects, nine stool specimens were required to establish the diagnosis.[2]

Webster discovered amebic infection in 147 cases with prior diagnoses of spastic colon, psychoneurosis, gall bladder disease, nervous indigestion, chronic appendicitis, and other diseases. Duration of symptoms varied from one week to over 30 years. In some cases, it took as many as six stool specimens to establish the diagnosis of amebiasis.[3]

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1. Cook, J.E., Briggs, G.W., and Hindley, F.W.: Chronic Amebiasis and the Need for a Diagnostic Profile, Am. Pract. and Dig. of Treat. *6*:1821 (Dec., 1955).

2. Rinehart, R.E., and Marcus, H.: Incidence of Amebiasis in Healthy Individuals, Clinic Patients and Those with Rheumatoid Arthritis, Northwest Med., *54*:708 (July, 1955).

3. Webster, B.H.: Amebiasis, a Disease of Multiple Manifestations, Am. Pract. and Dig. of Treat. *9*:897 (June, 1958).

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REFERENCES:

1-11. Papers read at Seventh Symposium on Antibiotics, Washington, D. C., November 4-6, 1959.

12. Phillips, F. M.: DECLOMYCIN—Seventh Interim Report. Department of Clinical Investigation, Lederle Laboratories, Pearl River, N. Y., December 4, 1959.

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genitourinary infection. Roberts, M. S.; Seneca, H., and Lattimer, J. K.,[1] New York, N. Y.—Ninety-one per cent of the Gram-positive and 27 per cent of the Gram-negative, among 66 organisms cultured from genitourinary infection, responded to DECLOMYCIN. Serum antibiotic activity was found three times greater than with tetracycline.

toleration. Boger, W. P., and Gavin, J. J.,[2] Norristown, Pennsylvania—Side effects with DECLOMYCIN were minimal. When dosage was 0.5 to 1 Gm. daily in divided doses, only two of 82 patients exhibited nausea.

activity level sustentation. Kunin, C. M.; Dornbush, A. C., and Finland, M.,[3] Boston, Massachusetts—Of the four tetracycline analogues, DECLOMYCIN Demethylchlortetracycline showed the longest sustained activity levels in the blood.

gonococcal infection. Marmell, M., and Prigot, A.,[4] New York, N. Y.—Of 63 cases of gonorrhea, 61 promptly responded after short courses of DECLOMYCIN. Therapeutic effect was found equal to that of intramuscular penicillin.

bronchopulmonary infection. Perry, D. M.; Hall, G. A., and Kirby, W. M. M.,[5] Seattle, Washington—Of 30 cases of acute bacterial pneumonia, all were afebrile following two to 10 days of treatment with DECLOMYCIN. Results were good in 21. . . . All of six patients with acute bronchitis responded promptly.

pediatric infection. Fujii, R.; Ichihashi, H.; Minamitani, M.; Konno, M., and Ishibashi, T.,[6] Tokyo, Japan—In 309 pediatric patients with various infections, DECLOMYCIN was effective in 75 per cent.

urogenital infection. Vineyard, J. P.; Hogan, J., and Sanford, J. P.,[7] Dallas, Texas—Clinical response in pyelonephritis correlated well with results of *in vitro* sensitivity tests, which showed some strains of *A. aerogenes*, Proteus and Pseudomonas more susceptible to DECLOMYCIN Demethylchlortetracycline than to its analogues.

pneumonia. Duke, C. J.; Katz, S., and Donohoe, R. F.,[8] Washington, D. C.—Results were satisfactory in all but two of 32 cases of acute bacterial pneumonia, of which only 11 were uncomplicated. No side effects were observed.

brucellosis. Chávez Max G.,[9] Mexico, D. F., Mexico—All of nine patients with *Br. melitensis* infection were afebrile after five days on DECLOMYCIN. Blood cultures were negative in all cases on the 20th day. Side effects were limited to slight temperature increases which abated in four days.

pustular dermatosis. Blau, S., and Kanof, N. B.,[10] New York, N. Y.—Results with DECLOMYCIN were excellent in both of two cases of impetigo, one of two cases of folliculitis, six of nine cases of furunculosis, all of three cases of acne rosacea and 26 of 45 cases of acne vulgaris. Overall, results were excellent or good in 85 per cent.

antibacterial spectrum. Finland, M.; Hirsch, H. A., and Kunin, C. M.,[11] Boston, Massachusetts—DECLOMYCIN Demethylchlortetracycline was found the most effective of the tetracycline analogues against two-thirds of 680 normally sensitive strains of 15 separate species.

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New Orleans, Louisiana

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Baltimore, Maryland

Murdina Desmond, M.D.
Houston, Texas

Ben Felson, M.D.
Cincinnati, Ohio

Michael DeBakey, M.D.
Houston, Texas

Schuyler Kohl, M.D.
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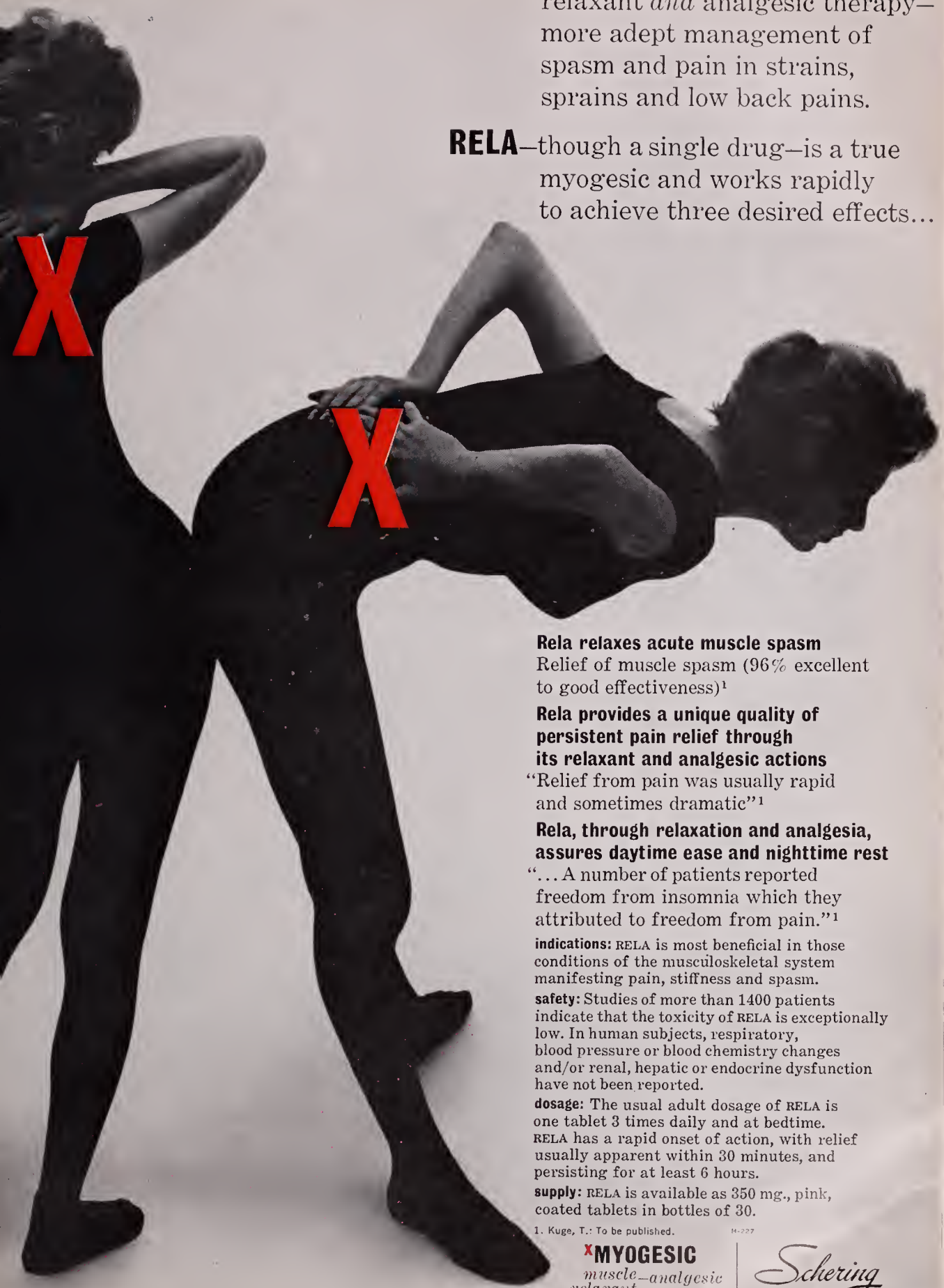
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1. Kuge, T.: To be published.

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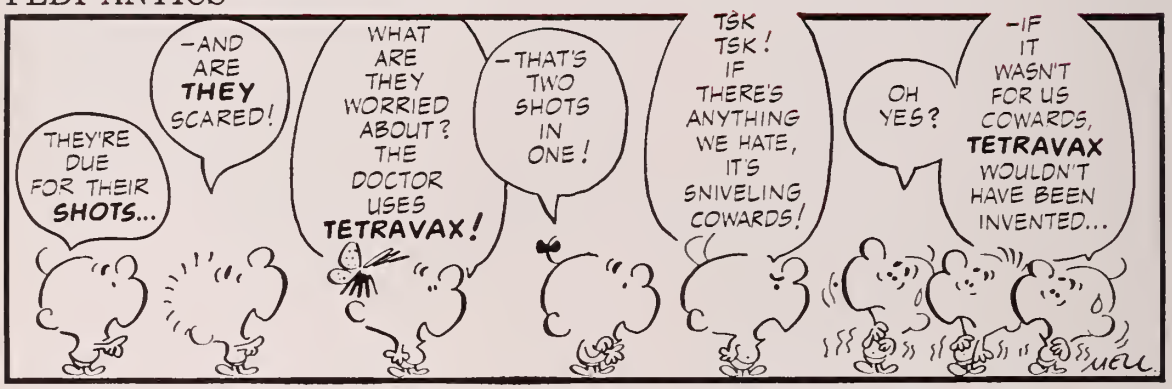
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Wein, A. B.: The Use of Carisoprodol in Orthopedic Surgery and Rehabilitation. Proceedings of the Symposium on The Pharmacology and Clinical Usefulness of Carisoprodol. Wayne State University Press, Detroit, 1959, p. 156.

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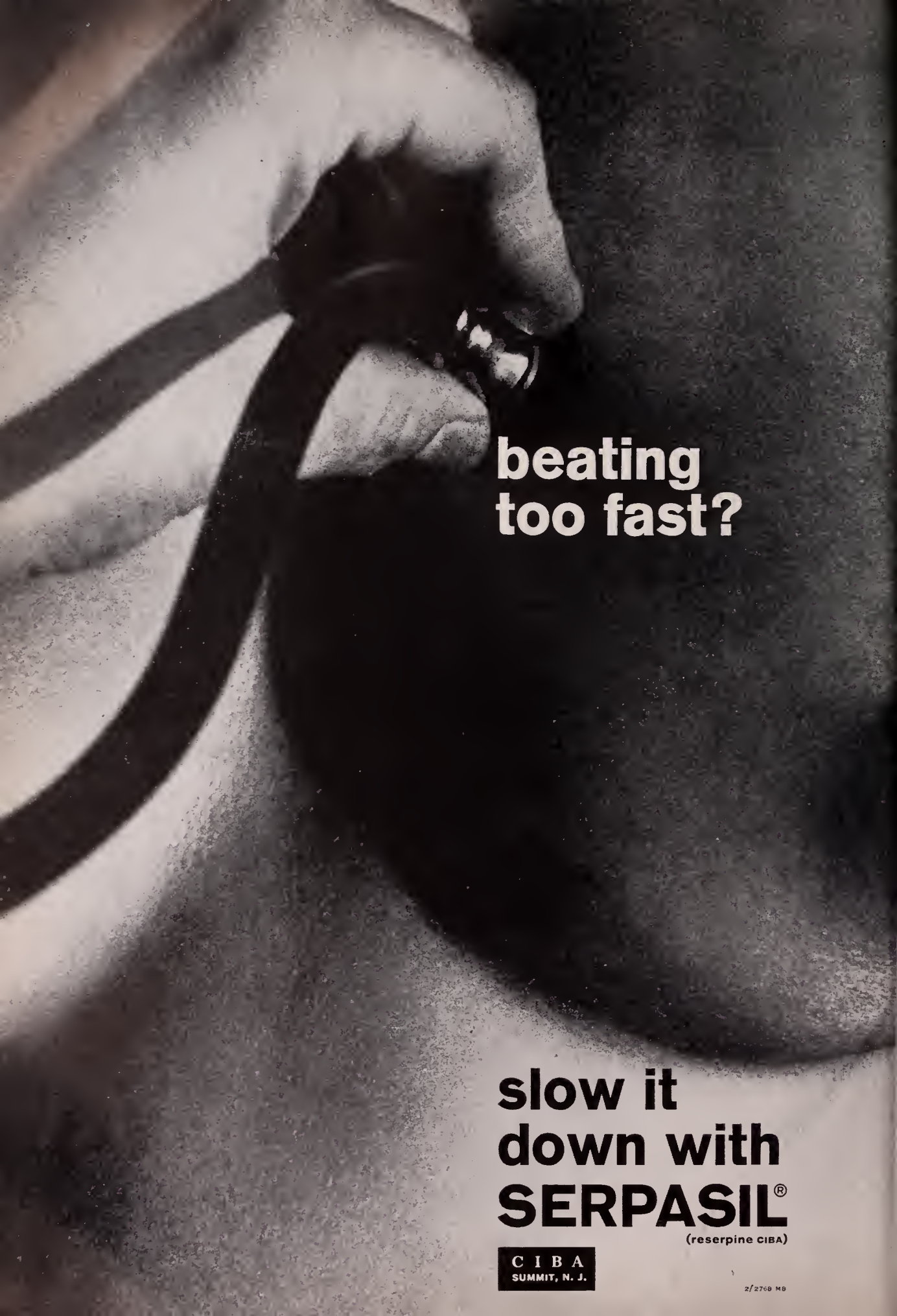
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Preferably, antibiotic therapy should be based on pretreatment culture of the offending pathogen, but in bacterial pneumonia the problem may well be too pressing to permit the required delay of 24 to 48 hours. A differential diagnosis among bacterial pneumonias, based on such clinical grounds as speed of onset, sepsis and pain may guide the choice of antibiotic for initiation of therapy.

Should clinical judgment dictate that antibiotic therapy be started immediately, at the same time a sputum sample or a subglottic swab can be sent to the laboratory for culture and sensitivity studies. If the response to the first antimicrobial agent proves unsatisfactory, a reasonable basis for changing therapy will then be at hand.

Choosing the Antibiotic

Since therapy must be started at once for bacterial pneumonia, it is advisable to choose a broad-spectrum antibiotic that quickly produces high levels of active agent (e.g., tetracycline phosphate complex, TETREX). Such an antibiotic probably has the best chance of controlling the pathogen, whether it be gram-negative or gram-positive. And if the laboratory report shows that the invading organism is much less sensitive to tetracycline than to other agents, the patient can then be changed to an appropriate antibiotic. If the difference in sensitivity is slight, then the possibility of side effects, sensitization, and toxicity should be evaluated before changing therapy to another antibiotic.

The greatest number of bacterial pneumonias are caused by pneumococci, which respond very well to penicillin, tetracycline, and chloramphenicol. Also, these antibiotics are usually effective against the other gram-positive coccal pneumonias. But penicillin is ineffective against the viral pneumonias and the gram-negative *Hemophilus influenzae* and *Klebsiella pneumoniae*. Although *K. pneumoniae* causes only about 1 to 2 per cent of pneumonia cases on the average,[1] these are apt to be acute and fulminating (Friedländer's pneumonia), with a high mortality rate if not effectively treated. Since pneumococcal pneumonia may be difficult to distinguish clinically from Friedländer's, except by gram-stained sputum smear, it may be wiser to start treatment with an agent also effective against Klebsiella.

Penicillin, however, in addition to having a limited spectrum, also causes many minor and some serious sensitivity reactions. In a recent survey[2] it was found that penicillin produced severe skin reaction. But most important was the observation that anaphylactic shock, with a fatality rate of about 9 per cent, was the most frequent serious reaction. Such severe reactions are almost always associated with parenteral administration.

Tetracycline is also clinically effective in primary atypical pneumonia.[3]

The tetracyclines (e.g., TETREX) have the advantage of a broad range of antimicrobial activity and low toxicity. And in addition, the physician does not have to trouble himself or his patients with repeated blood studies when he prescribes TETREX. Minor reactions such as gastric upsets or mild skin rashes occur occasionally. The most serious side effects are staphylococcal and monilial overgrowth, but these are rare and can be adequately controlled.

No one would deny that appropriate antibiotic therapy has greatly reduced morbidity and saved many lives of patients with bacterial pneumonia. Nevertheless, general supportive measures in the care of patients remain important even today. Especially in the desperately ill patient, antibiotics are not considered as substitutes for the individual evaluation, clinical observation and judgment of the physician.

Some Micro-organisms Susceptible[a] *to Tetracycline* (TETREX)[b]

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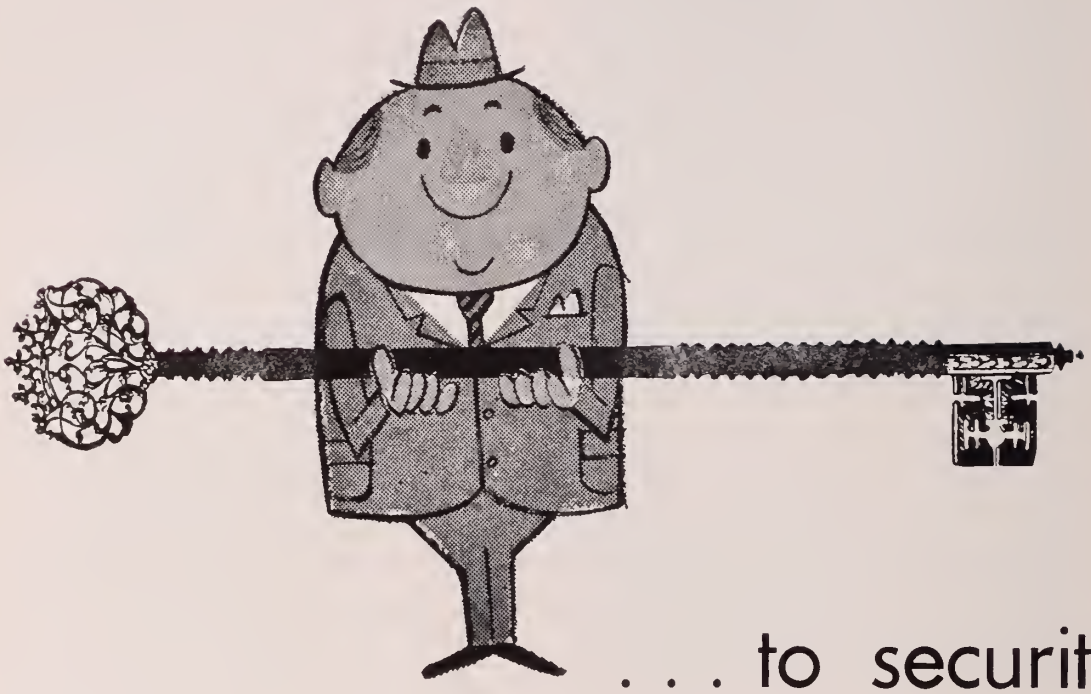
a Some strains are not susceptible.

b Table adapted from Goodman, L. S., and Gilman, A.: The Pharmaceutical Basis of Therapeutics. 2nd edition, New York, The Macmillan Co., 1956, pp. 1322-1323.

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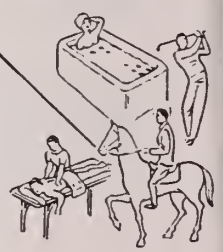
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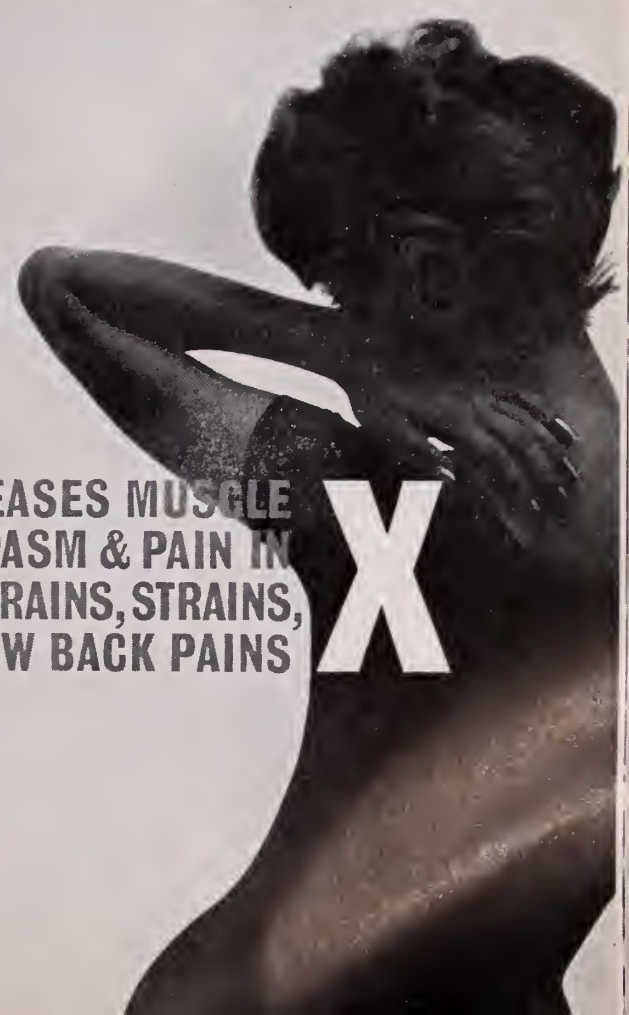
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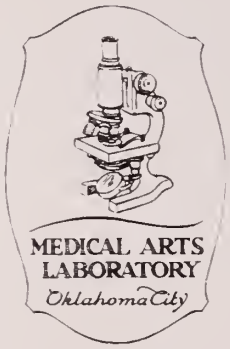


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Index to Advertisers

Abbott Laboratories xx
Ames Company, Inc. ..... inside back
Arlington Hotel liv

Beverly Hills Clinic lxv
Bone and Joint Hospital lix
Bristol Laboratories, Inc. ..... xxxiv-xxxvii, insert, li
Burroughs-Wellcome & Company, Inc. ..... insert

Ciba ..... x, xlviii
Coca Cola Bottlers lv
Corn Products Refining Co. ..... vi and vii
Coyne Campbell Sanitarium lix
Credit Service liii

Geigy xv
General Electric xvi
Goldfain Laboratory lx

J. E. Hanger l

Drs. Lain, Lamb and Jones lx
Lederle Laboratories ..... viii and ix, xxi, xxvi, xxxi, xl and xli, l, liv, lvi, lxv
Eli Lilly & Company xiv

Medical Arts Laboratory lviii
S. E. Massengill Company xxxix

Melton Co., Inc. lvi
Merck Sharp and Dohme, Inc. ..... iv, xxx, xlvi
McAlester Clinic lxi
Midwest Surgical Supply Co., Inc. lxv

The Neurological Hospital lxiv
North American Accident Insurance Co. lii

Oklahoma Allergy Clinic lxi
Osler Radioisotope Laboratory lxiv

Parke Davis and Company ..... inside front and iii
Pfizer Laboratories ..... xi, xxiii, xxxviii
Physicians Casualty & Health Assn. liii
Physicians Laboratory lxiv
Pitman-Moore Company xxii

A. H. Robins Co., Inc. xlix
Rock Haven Hospital lxiv
Roerig ..... xxv, xxxii and xxxiii

St. Paul Companies lvii
Sardeau, Inc. xvii
G. D. Searle & Company ..... xii and xiii
Schering Corporation ..... xlii, xliv and xlv, l, liii, lv
Seven-Up Bottlers lvi
Smith-Dorsey ..... xviii, xxiv, xxvii
Smith, Kline & French Laboratories ..... back cover
Springer Clinic lxii
E. R. Squibb and Sons xliii
Stout Clinical Laboratory lxii
Sugg Clinic lxiii

Terrell's Laboratories lxiv
Tulsa Clinic lxiii
S. J. Tutag & Co. lvii

The Upjohn Company ..... xxviii and xxix

Wallace Laboratories ..... insert, xlvii
Winthrop Laboratories ..... xix, insert

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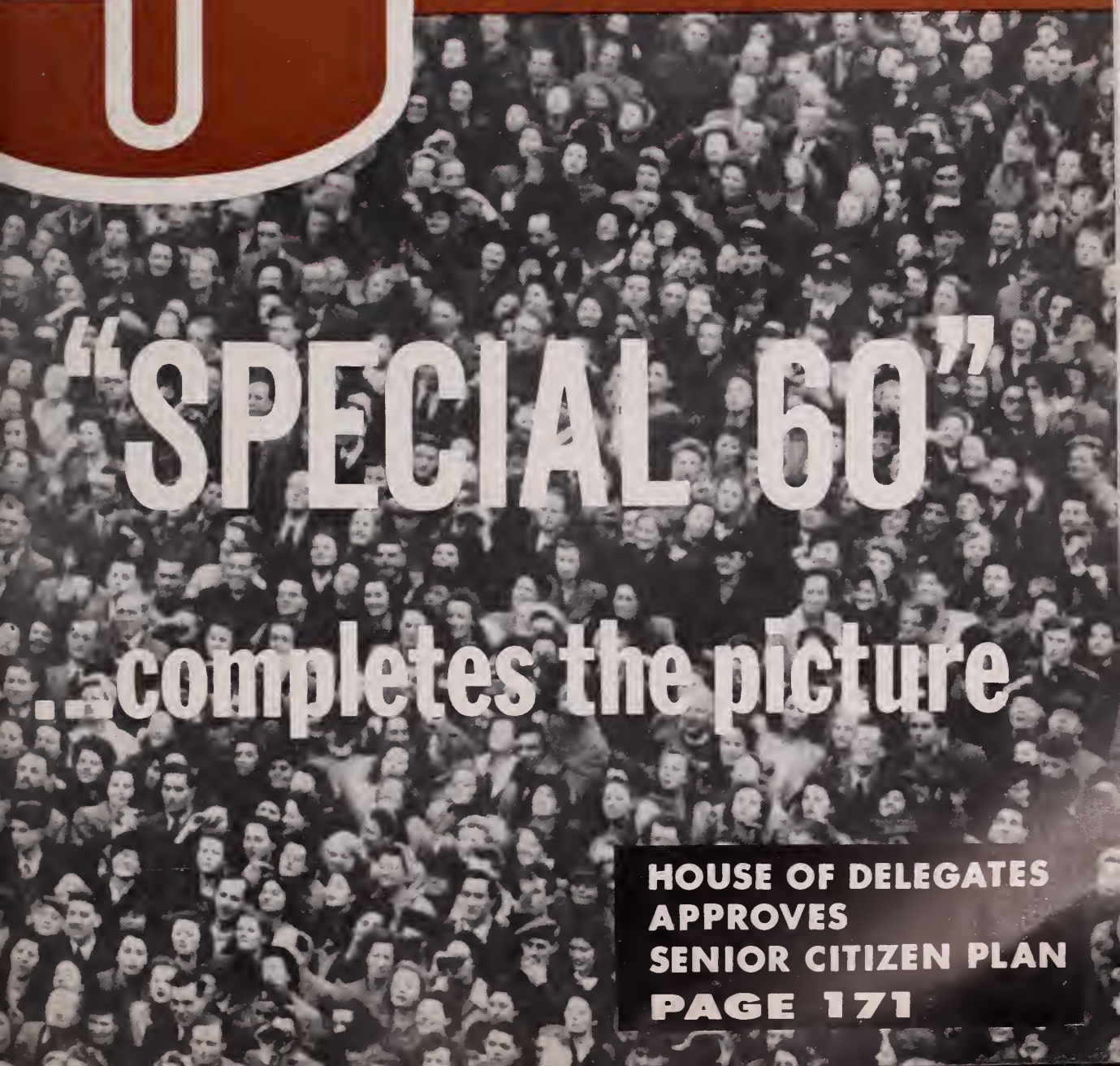
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CONTENTS

EDITORIALS

Dr. Isabel Cobb, Her Times and Ours 129
President's Letter 158

SCIENTIFIC ARTICLES

Children's Eyes, *Tullos O. Coston, M.D.* 131
A Clinical Study of Meprobamates Used as a Muscle Relaxant Compound, *William N. Harsha, M.D.* . . . 135
Resections for Tuberculosis at Eastern Oklahoma Tuberculosis Sanatorium, (A Four Year Experience), *John M. Carey, M.D., Frank L. Bradley, M.D., Forrest P. Baker, M.D. and Edna Colbaugh* 138
A Confirmed Hospital Epidemic of Asian Influenza, *Harold G. Muchmore, M.D., Frances G. Felton, M.D., and L. Vernon Scott, Sc.D.* 142

MEDICAL CENTER

Message from the Dean 146
Medical Statistics: Its Concepts and Uses, *Edward N. Brandt, Jr., M.S.* 147
Enteroliths As a Cause of Intestinal Obstruction, *John L. Ritan, M.D. and Ira Ross Grimes, M.D.* . . . 153
Regents Appoint Four New Faculty Members 155
Schottstaedt to Head Department of PM and PH . . 155
Abstracts 156

SPECIAL ARTICLE

A March to Utopia—True or Political, *Ardell T. Everett* 160
The Sixties . . . Decade of Decisions 165
Factors Relating to Physician Distribution in Oklahoma, 1950-1959, *Robert C. Lowe, M.D., James A. Hagans, M.D. and Pearl Fisher, Ph.D.* 168

NEWS

Indemnity Program for Senior Citizens Approved . . 171
Kennedy, Liberals Push for Forand-Type Legislation . 172
Physicians Play Leading Role in Aged Study 174
'Something for Everyone' At Annual Meeting 175
Program Completed for Third Oklahoma Colloquy . . 178
Medicare Identification System Outlined 180
Southwest Allergy Forum Will Meet in Oklahoma City . 181
Federal Health Act; A Significant Step 183
Coming Meetings 187
Auxiliary News 192
Twenty-five Years Ago 193
Physician Placement 195
Miscellaneous Ads 197, Index to Advertisers lxxvi

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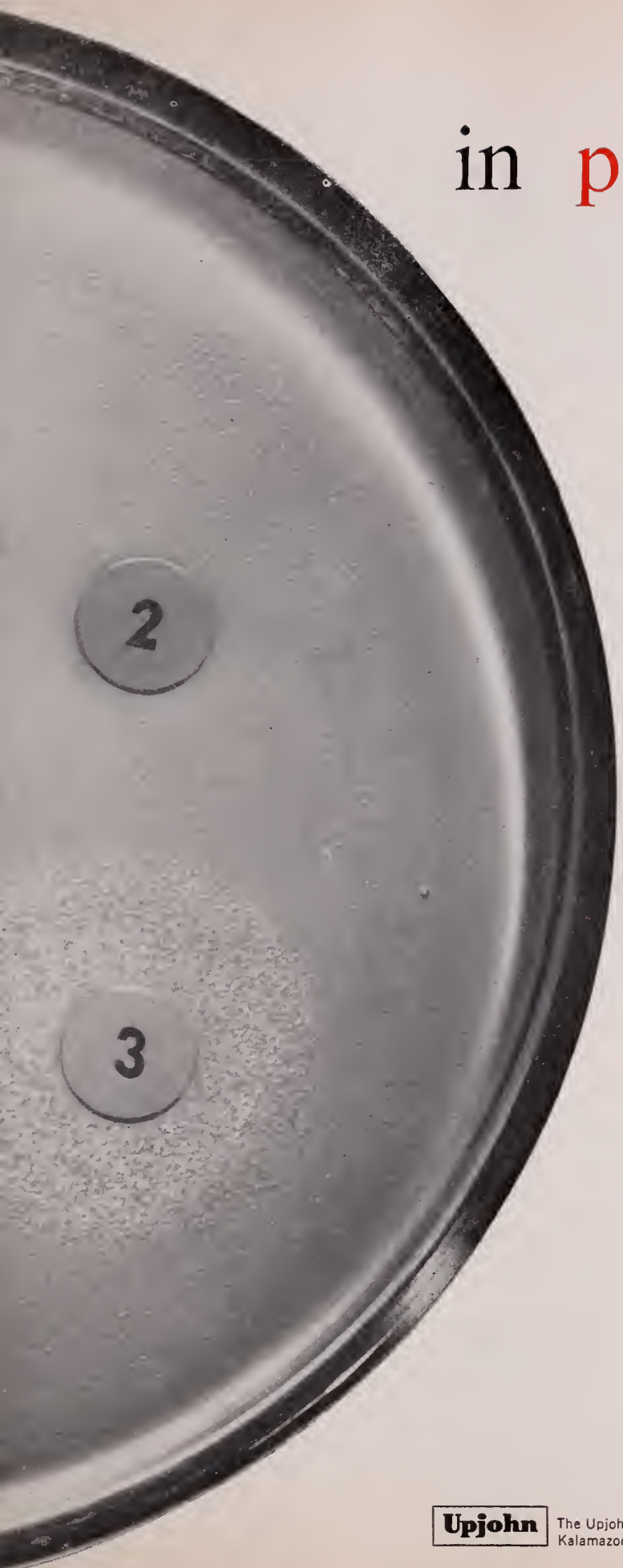
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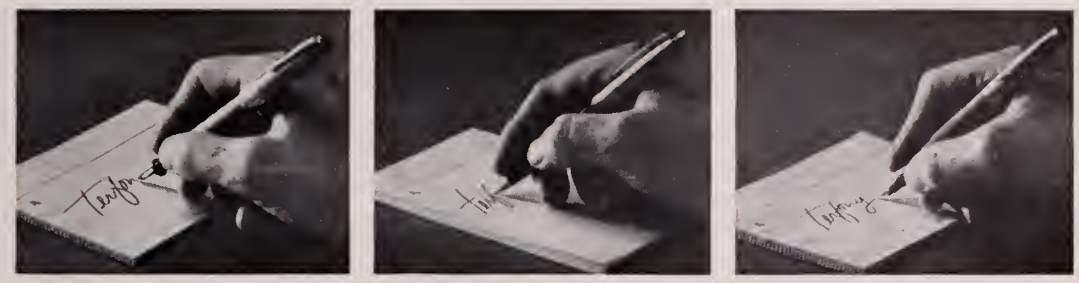
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1. Projection from Vital Statistics, U.S. Government Dept. HEW, Vol. 48, No. 14, 1958, p. 398.
2. Modell, W.: Drugs of Choice 1958-1959, St. Louis, C. V. Mosby Company, 1958, p. 347.

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Editorial

Dr. Isabel Cobb
Her Times and Ours



On its way to the archives at Norman, I had a chance to go over the daily log of an Aunt of Doctor Forrest Etter of Bartlesville. She was born, Isabel Cobb, in Cleveland, Tennessee in 1858 and came to Indian Territory in 1870 at the age of 12. After graduating from Female Seminary in Tahlequah she taught school for a short time before entering college in Glendale, Ohio in preparation for Medical School. She graduated from Woman's Medical College in Philadelphia in 1891 and then had a two year internship in State Island Children's Hospital. She came home to practice five miles southeast of Wagoner from 1893 to 1930.

Doctor Bell Cobb's record book begins in March 1893. The year 1895 yielded $326.10 for her. Those were, however, real dollars, not inflated dollars. The next year she collected $274.00. She wrote in a clear concise legible way—giving for each patient visit the history, her findings and treatment and the charge, if any. The notes are brief and to the point but managed to convey the love of people that must have endeared her to all her patients as well as the whole community. The story of one patient taken from her log reflects a much better image than I can draw.

Mary Harvey got sick on Thursday, Nov. 26, 1896. She came to see Dr. Bell on Sat. 28th and was given some medicine for the

fever. On her return Nov. 30, something was added for restlessness. On Dec. 1, she was about the same and was given medicine for worms. The next day, Dr. Bell went out to see her and made this note:

Dec. 2—"Temp. 102.5, sleeps, moans some on expiration, coughs but little, pulse bounding, respiration normal, little tenderness over liver and in right iliac region, one spot resembling typhoid, tongue dry at first, is now moist and clear, is thirsty, eats nothing, lips sore."

Dec. 3—"No change—coughs a little more and looser. No worms passed—appetite better—drank some milk last night."

Dec. 4—"Mouth quite sore—Temp. 102 (5 P.M.). Seemed much better this morning, sweated and seemed to have no fever at all."

Dec. 7—"Thinks she is doing well—no fever since Friday night."

Dec. 8—2 A.M.—"Much worse—rt. lung begin. involv. but thought might be hypostatic congestion—took Dr. Braziel out in P.M. — lung involvement increased — pneumonia."

Dec. 9. "Rested well at night—no improvement."

Dec. 10 "Died at 2 A.M."

Doctor Bell's bill for looking after Mary Harvey in her final illness was $8.00. Her book contained this note beside the charge—"Dr. B. charged $13.00 hence my small bill."

There were many Mary Harveys in those days. There were many still births; many new borns who lived only a few days or weeks; women in labor at home in the country one or two days with the doctor staying there; lots of mud; horses to feed and water; wood stoves; out door biffies, winter and summer; oil lamps; a well or spring; straw mattresses; child-bed fever; membranous croup; worm medicine; opium; purgatives; poulties and fomentations.

Those were the good old days when a dollar *was* a dollar. Our thinking is cockeyed —what we really want to do is to buy with today's dollars at yesterday's prices. This is what frustrates the average guy about inflation—he can't.

It is not clear to me when inflation would become a serious threat to the country's monetary system, but no less a threat would be the millions now making things who could not be hired—for yesterday's dollars would not be numerous enough to buy these things. The dollar is only the medium of exchange of man's labor which is the basic point of departure. When I was a boy, my father was a "concrete contractor." He built sidewalks, driveways, steps, porches, foundations, silos, small culverts and the like. His crew were stalwart negro men, many with large families. The day laborer's pay was 10c an hour—$6.00 on Saturday night for six ten-hour days. It is true there were no deductions for social security—no sales tax—no withholding. Dr. Bell, bless her, can have her real dollar, her snuff, her privy, her mud, her santonin and calomel, her Blands pills, Dover's powders, her mustard plasters and her pleasant buggy rides through the air-clean country-side. As for me, I'll take the plumbing with $5.00 an hour for the plumber, paved roads and streets, sales tax, cigarette tax, income tax, hidden tax, luxury tax, social security, automobiles that fog the air with exhaust, penicillin, the sulfa drugs, blood transfusions, good anesthesia, sterile techniques, and a dollar whose value is said to be depreciated.

"Backward, turn backward, O Time, in your flight." Nuts.—*B.H.N.*

ATTENTION COUNTY SOCIETY OFFICERS!

- House of Delegates, May 1, 1960
- Annual Meeting, May 2, 3, 4, 1960
- County Resolutions should be mailed to OSMA office soon!
- Names of new Delegates and Alternates have been due since January 1, 1960!

Scientific Articles

What a practitioner should know about strabismus, injuries, retrolental fibrophasia, and vision tests.

CHILDREN'S EYES *

TULLOS O. COSTON, M.D.

Since his graduation from Johns Hopkins University in 1930, Tullos O. Coston, M.D., has been certified by the American Board of Ophthalmology. In addition to his private practice, Doctor Coston is Clinical Professor of Ophthalmology at the University of Oklahoma School of Medicine.

Doctor Coston is a member of the American Academy of Opthalmology and Otolaryngology, the Johns Hopkins Surgical and Medical Society, the Wilmer Residents Society and the Pan-American Association of Ophthalmology.

I WAS ASKED to discuss with you the subjects of strabismus, injuries to the eyes, visual testing, and retrolental fibroplasia. In the allotted time, I shall be able to devote about seven minutes to each one of these subjects. Obviously, I shall attempt to dwell briefly on the points of interest and importance to the pediatrician and the general practitioner.

Strabismus

The infant's eyes should be straight by three months of age. Any deviation of the visual axes beyond that age almost certainly is indicative of a muscle imbalance or a lack of vision in one or both eyes. The pupils should be widely-dilated with 5% Homatropine in a 1% solution of Paredrine and the fundi examined very carefully. Not infrequently, congenital cataract, retinal scars, malformations of the inner eye, or the dreaded malignant retinoblastoma may be found. The last-named condition can often be treated quite successfully if discovered during the first four to six months of the infant's life.

If the ocular structures are found to be normal, it is usually wise to defer the treatment of the strabismus until the child is approximately eighteen months of age. At that time, atropine ointment, 1%, should be used in each eye three times daily for three days and the refractive power of the eyes determined by retinoscopy. This can be done only by the ophthalmologist. The non-medical practitioner, an optometrist, cannot or should not use drugs in the eye. If atropine is not used, as indicated above, the accommodative mechanism of the eyes remains active and it is as impossible to measure the refractive power as it would be to measure the length of an accordion while it is being played. Atropine, and scopolamine, the other drugs used for this purpose, can be very dangerous and should not be placed in the hands of non-medical practitioners by well-mean-

*Presented at the Clinical Session of the American Medical Association, December 1-4, 1959, Dallas, Texas.

ing but misguided physicians. This warning applies, also, to the various mydriatics used to facilitate the study of the fundus.

It is important to know the refractive power of the eyes because many cases of strabismus with high hyperopia can be corrected by the wearing of glasses. These may be prescribed as early as eighteen months of age.

Eye exercises are helpful in, perhaps, fifteen to twenty per cent of cases of strabismus. They are of little value in divergent strabismus. Such exercises cannot be started, usually, before the child is three to three and one-half years of age. Such training should be directly under the ophthalmologist or the orthoptic technician. The last-named works under the direction of the ophthalmologist and is usually located in a teaching-medical center. This type of service is not available in many areas.

These exercises attempt to train the eyes to work as a team and to stimulate fusion of the images of the two eyes in the visual center of the brain. Most important, also, is the matter of developing equal vision in the two eyes. The crossing eye usually has low vision and this must be stimulated. This is done by occluding the eye with better vision and forcing the use of the low vision eye. If this is not done before five years of age, improvement in acuity of the amblyopic eye often does not occur.

The third method of treating strabismus is that of operation. There is no set age at which the various operative procedures should be carried out. In general, operative correction should be completed before the child enters school. In some cases, operations should be done as early as eighteen to twenty-four months of age. In most instances, the combination of the wearing of proper glasses, eye exercises, and operative procedures are necessary. In many cases, more than one operation is needed and almost without exception, in all cases the eyes can be made straight by using one or more of the methods discussed above.

Tests for Visual Acuity

In general, a child must be three to three and one-half years of age before any accurate visual acuity examination can be done. During the first month or two of life, many normal infants will not appear to notice a light. By the time the child is three months of age, the eyes should turn toward a strong light stimulus, such as a flashlight. By the age of six months, a child will grasp or attempt to grasp small objects when offered. The infant between twelve and eighteen months of age may be tested by placing a small white bead in the palm of the hand. With normal vision, many infants will attempt to pick this up. At the age of three, the child may not call out the names of pictures on one of the regular Snellen test charts but will name, across the room, such objects as a small wrist watch, a pencil, a key, a safety pin or a penny.

Children who are old enough to cooperate on the Snellen charts can be tested with the picture charts at ten or twenty feet or with the so-called "E" charts. On the latter, the block letter "E" lies in various positions and the child is given the same letter cut out of cardboard to hold in his hand. He is asked to hold his "E" in the same position as the "E" being pointed to on the test chart by the examiner. This same test can be used with a close up "E" card. The "E" card testing is usually used until the child is in the first or second grade because many children do not know the forms of the alphabet before that age.

The Harrington near-test chart, showing pictures of familiar objects on one side and the letters of the alphabet on the other, is an excellent chart. All of these may be obtained from any reputable optical company. It is important to know that a child usually does not obtain his or her full visual acuity until the age of nine years. An acuity of 20/40 or 20/50 may be quite normal for a child between the age of three and five years.

Injuries

The most common injury to the eye is a corneal abrasion and it is usually impossible to detect this without the aid of fluorescein staining. This is best done by placing a couple of drops of sterile saline or boric acid solution on the end of a sterile fluorescein strip and allowing a drop to fall into the lower cul-de-sac by pulling the lower lid

down. Fluorescein solution becomes contaminated very quickly and is often the source of very virulent ocular infections. Let me urge you to use the sterile paper strips impregnated with fluorescein. As you know, a corneal abrasion will stain green after fluorescein has been instilled.

Any foreign body on the cornea should be removed and, unless it is embedded, this can be easily done by using the side or point of a sterile hypodermic needle, after the instillation of a local anesthetic such as Dorsacain or Pontocain, ophthalmic solution. An antibiotic ointment should be instilled and a pad placed on the eye. The eye should be re-examined the followig day for detection of any possible infection. It is most important to wear a magnifying loop and have a good source of light in detecting and removing the foreign bodies from the eye.

If, upon examining the eye, one sees a distortion of the pupil, hemorrhage in the anterior chamber, a black reflex in the pupillary space (with the ophthalmoscope) instead of a red one, a collapsed anterior chamber, or prolapse of iris or uvel tissue, the patient should be sent immediately to the nearest ophthalmologist and nothing done to the eye except to place an eye pad over it. If the patient cannot be placed under special care for some hours, it is well to give a parenteral injection of penicillin.

Retrolental Fibroplasia

This disorder of the eye, which first appeared about eighteen years ago with the advent of closed incubators for premature infants, is caused by the uncontrolled administration of oxygen. Over a period of twelve years before the cause was determined, eight thousand babies lost their vision in the United States. Now that the cause is known, very, very few additional cases should ever occur.

Retrolental fibroplasia has been reported in a few, rare instances in full-term infants. It is to be considered, however, a disease of prematurity. It rarely occurs in infants weighing four pounds or more and is most likely to occur in infants weighing three and one-half pounds or less. The disease has been produced in several species of animals by administration of oxygen.

The first objective finding is a dilatation of the retinal blood vessels. This occurs after the infant has been removed from the higher oxygen concentration and may appear within a matter of hours or days. The peripheral areas of the retina then become grayish, hazy, and edematous in appearance. This is followed in a matter of seven days to two weeks by the growth of new retinal vessels which grow in such wild fashion as to come forward into the vitreous, break, and bleed. The resulting vitreous hemorrhages and new vessels stimulate the production of glial tissue bands, which, in turn, tug at the retina and detach it. The final stage is one of a glial sheet, containing blood vessels and retina, located behind the lens. Many cases do not progress to this final stage but are arrested spontaneously at some point before this, leaving the child some vision. Retrolental fibroplasia never develops after the age of three months.

How can this disease be prevented? First of all, do not administer oxygen to the premature infant unless it is absolutely necessary to do so. In other words, be certain that positive signs of oxygen want are present.

Secondly, never allow the oxygen concentration in the closed incubator to go above forty per cent; preferably, it should be kept around thirty per cent. It is most important to use an oxygen analyzer and have the oxygen concentration sampled every four to six hours. Any nurse can be trained to use the oxygen analyzer and she should be instructed to keep a chart of the oxygen concentration, just as she maintains the temperature chart.

Thirdly, shorten the time of oxygen administration as much as possible. The development of retrolental fibroplasia is in direct proportion to the concentration of oxygen and the duration of time that it is administered.

I wish to speak a word concerning the medico-legal aspects of retrolental fibroplasia. It is most important to sign a statement on the patient's chart that the admin-

istration of oxygen was necessary to preserve the life of the infant. This should bear the attending physician's own signature. It is most important to be able to prove, even years later, that the concentration of oxygen was maintained between thirty and forty per cent and was not allowed to go above the latter figure. The records must be carefully preserved since legal action can be taken by the infant upon reaching the age of twenty-one years.

There is no treatment for retrolental fibroplasia except, perhaps, in one phase of the disease. Let me stress the fact that it is of no value to place the infant back in oxygen if the disease begins to develop. It was thought that this might be helpful at one time in the past, but this did not prove to be true.

At the stage of the disease, characterized by beginning retinal detachment, an operative procedure for reattachment of the retina may preserve a great deal of vision. I have been fortunate in being able to preserve vision in one such patient, the only case in which this has ever been attempted to my knowledge. In order to carry out such a procedure, one must be observing the fundi almost daily as the disease progresses, because the favorable time for such an operation would be no longer than seven to ten days at most.

My hope is that more such patients might have the advantage of such a procedure. It would be wise to have the fundi of premature infants weighing three and one-half pounds or less (if placed in oxygen) examined by an ophthalmologist, weekly, upon removal from the higher oxygen concentration. The appropriate cases for treatment might thus be found.

Summary

It has been said that if one new idea is gained from the presentation of a paper, the time has been well-spent. I wish to reemphasize these points:

(1) The importance of early medical examination of the strabismic infant.

(2) The proper technique of corneal staining with sterile fluorescein strips.

(3) The limited use of low concentration oxygen for the premature and the medico-legal dangers attendant thereto.

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An attempt to evaluate acute skeletal muscle relaxing effects of meprobamates in treatment of orthopedic patients. The drug may be of real value in the management of patients whose discomfort is largely due to muscle spasm.

A Clinical Study of Meprobamates Used as a Muscle Relaxant Compound

WILLIAM N. HARSHA, M.D.

William N. Harsha, M.D., graduated from the University of Kansas School of Medicine in 1948. He has been certified by the American Board of Orthopedic Surgery and in addition to his private practice, is Clinical Assistant Professor of Orthopedic and Fracture Surgery at the University of Oklahoma School of Medicine and instructor in the College of Law at the University of Oklahoma.

Doctor Harsha is a member of the Christian-Medical Society and the American Rheumatism Society.

COMPLAINTS about the low back and the neck are common everyday problems for the general practitioner as well as the orthopedist. In most of these patients pain is largely due to muscle spasm. The spasm itself is in response to some underlying injury or abnormality. The voluntary muscles of the low back and neck are oriented to go into reflex spasm when underlying tissues are injured or diseased. In the acute cases muscle spasm generally has its origin in an injury, allbeit a trivial one at times. There is ligamentous stressing or even tearing of a few fibers and the muscle spasm is an attempt to protect these areas from further insult until healing has occurred. So-called whiplash injury seen after rear-end auto collision is a notable example of this entity. All gradations of pain and disability may occur. The acute ruptured intervertebral disc in the low back has as one of its principle disabilities muscle spasm to splint this area. In the arthritic patients who twist, strain, sneeze, or pick something off the floor in an unfortuitous position ligamentous strain with reflex muscle spasm is produced.

The author is the first to admit that all backaches are not symptomatic dominently on the basis of muscle spasm yet a significant component of pain in the back and the neck is on this basis. The rational treatment of these complaints, in part, always includes relief of muscle spasm. This can be accomplished by the well-known modalities of deep heat, massage, splinting, traction and of restriction of activity to prevent re-injury. In the very acute episode of these modalities are not uniformly effective, or at least not totally so. The use of narcotics can be had and substantial relief of the pain will result in most cases yet there are many reasons to interdict the regular and sometimes rather prolonged use of narcotic agents. This is particularly true in the ambulatory patient. There has been a continued search through the years for other medicinal agents that have the ability to produce voluntary muscle relaxation and at the same time treat the patient with the other modalities that will prevent re-injury, giving the patient a satisfying and sound regime of therapy. There are many agents known to block muscle-nerve reflex pathways, either within the central nervous system or at the muscle-nerve end plates. The bulk of these carry inherent risk to respiratory function and require very close supervision. The curare series of drugs are the dominent ones in this type of therapy. Also in common use

are the chemical derivities, zoxazolamine, the methocarbaminols, and the mephensesin compounds. The latter group of chemicals in the hands of many workers have perhaps been the most successful but in themselves have undesirable side effects. Several investigators have used meprobamate compounds to aid in relaxation of voluntary muscle tension or spasm.[1,2,3] Vazuka conducted a comparative study with the effects of muscle relaxant compounds Robaxin, Equanil, and Flexin and found Equanil in adequate doses to be the more effective.[3] Dickel and her co-workers were able to demonstrate by electromyographic studies in patients treated with meprobamates that relaxation of voluntary muscles was effected with greater ease and that there was increase in muscular coordination.[4] Smith and his co-workers found that meprobamate compounds were to be relatively long acting as opposed to the mephenesin type compounds and that the peak effect frequently can be reached from thirty to sixty minutes.[5,6]

Study

At the outset it was undertaken to get patients whose injury as regards type, severity and control of muscle spasm would be reproduced in many others. They should be in a controlled environment so that exogenous influences of either medicinal agents or undue physiotherapeutic modalities would not influence the opinions of examiners. At best this cannot be a precise type of investigation because it relies mostly on the subjective reliability of the patient and to a lesser extent of the muscles involved. By having the same examiner examine patients repeatedly, establish a real element of rapport with the patient and attempt to understand the problem with the patient and explain to the patient what is to be accomplished, a rather reliable evaluation of the effects of therapeutic agents can be had.

The type of patients included in this report are those with acute painful states to their neck and to their low back following trauma. A deliberate attempt was made to exclude patients with demonstrable arthritic changes, either clinically or by x-ray. A like attempt was made to exclude the patient who might expect some financial gain through workmen's compensation or other type of insurance coverage. A total of thirty-two acute painful necks and forty-eight acute low back problems, some with, others without sciatica were studied. Sixteen of this group were deleted during the course of study because problems of litigation were noted.

Measurement

The orthopedic evaluation of the neck problem included a precise and adequate history and physical examination along with x-ray studies. The ranges of motion were recorded and a deliberate attempt to isolate areas of distinct muscle spasm, particularly an evaluation of the trapezius, the levator scapulae or the posterior erector spinae muscle groups. In a similar fashion, for the low back complaints, a thorough history and orthopedic examination of the low back, including x-ray examination was carried out and again an attempt to isolate specific muscle groups involved was made, principally sacro-spinalis and erector spinae muscle groups in this area.

Patient Selection

Two types of patients were treated. Thirty-four patients were treated on an ambulatory basis, and thirty were hospitalized with their acute episode. Sixteen others initially started were considered unsatisfactory for this report due to litigation. It was explained to the patient that they were receiving one type of medicine only to control their pain. The patients then were placed on meprobamate (Equanil) in doses from 400 to 800 mgs. q. i d. for the first three to four days then tapering down to doses of 200 to 400 mgs. for an additional eight to ten days. The patients were checked at 48 hour intervals or more often as seemed indicated and evaluation of the muscle spasm, limitation of range of motion and subjective complaints were made. Other therapeutic modalities did include bed rest, local heat in the form of heating pad and proper posturing. Head pillows were removed with the use of a roll underneath the neck in case of cervical complaints and the use of a flexion position in bed in cases of lumbar complaints. In the case of hospitalized patients

in more severe distress larger doses of Equanil were used. At times, 1,000 to 1,200 mgs. on a q. i. d. basis being used but in cases where larger doses were used the smaller 400-800 mg. q. i. d. schedule was first followed and then if effect was not noted the dose was increased. In some of the patients placebo tablets were used. These appeared grossly to be Equanil tablets and were substituted without the patient's knowledge and changes of symptoms and signs, if any, were noted.

Results

For the group of ambulatory patients relief of the painful component of muscle spasm or tension and increased sense of well being were frequently produced. The effect was considered to be good in 70% of the cases, fair in 20% and apparently offered no change in 10%. The criteria for good was complete comfort so that bending and twisting activities could be resumed without distress. Fair was considered to be improvement of pain and spasm but continued awareness of discomfort in the affected area and no change when the pain and discomfort or muscle spasm was the same or apparently worse. In the case of the hospitalized patients good results were achieved using meprobamate in 78% of the cases. Fair results were achieved in 16% of the cases and poor or unsuccessful in 6%. No patient was encountered during the hospitalization in which acute muscle spasm demonstrable by palpable changes and of subjective complaints in predictable areas could not be relieved if large enough doses of meprobamate was used. On three cases of rugged working type of individuals with low back complaints, 1,200 mgs. of Equanil four times daily was necessary for a three to four day period.

The promptness of relief if a therapeutic effect on muscle spasm was obtained varied substantially. It generally occurred within 36 to 48 hours, and recurrence of a spastic muscle state followed the last dose or the introduction of placebo medication instead of the meprobamate within 12 to 24 hours.

Because the patient with acute muscle spasm does not ordinarily have severe prolonged disability or underlying organic disease relief was usually permanent once it had been achieved for a period of from five to ten days. Chronic, longer lasting painful states in a second group were studied in a cursory fashion. In these demonstrable muscle spasm was either absent or equivocally present. The use of meprobamate was not so outstanding in its pain relief ability. However, when coupled with standard analgesic agents such as 10 to 15 grains of aspirin with each 400 mgs. dose there was a noticeable improvement in its efficacy.

Summary

Reviews of a total of 64 patients with acute spastic phenomena of an orthopedic nature have been studied by use of meprobamate as a specific muscle relaxant. The results for the acute episode are quite promising. Earlier and more comfortable rehabilitation was possible, particularly when exercise regimes were indicated. In many of the cases it also appeared that there was a close relationship between the relief of anxiety and the relief of discomfort. A sense of well being seemed to minimize the tendency to complain about the initial acute problem. The results for the more protracted chronic types of musculo-fascial pain were less promising unless combined with analgesic agents. Side effects noted with the use of meprobamate were slight. One case of urinary retention followed the use of 800 mgs. of Equanil q. i. d. for two days which was relieved promptly by substituting a placebo but promptly re-occurred on two subsequent occasions when Equanil was used. One case of dermatitis medicamentosa was encountered which responded promptly to antihistaminic medication.

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411 N.W. 11th, Oklahoma City, Oklahoma

A Four Years Experience

Resections for Tuberculosis at Eastern Oklahoma Tuberculosis Sanitorium

JOHN M. CAREY, M.D.
Thoracic Surgeon

FRANK L. BRADLEY, M.D.
Assistant Superintendent, Eastern Oklahoma Tuberculosis Sanatorium

FORREST P. BAKER, M.D.
Superintendent, Eastern Oklahoma Tuberculosis Sanatorium

EDNA COLBAUGH
Administrative Assistant

Introduction

IN APRIL, 1955, a program of pulmonary resections for tuberculosis was initiated at the Eastern Oklahoma State Tuberculosis Sanatorium. Until April, 1959, 71 patients have had pulmonary resections. This paper presents the preliminary results of this four years experience allowing minimal follow-up of six months. This report is of interest because it presents the results of a 300 bed institution in which a resection program has been recently initiated.

Material

Table I presents the clinical data on these 71 patients. Since this report presents the results of our initial resection program, it may be easily discerned that many of the patients had exhausted the benefits of a medical regimen prior to operation. Sixty per cent of these patients had at least one prior admission to this sanatorium. An indeterminate number had therapy at other institutions or from their home physicians. The most striking feature of this group was that 83 per cent of the patients were still sputum positive with open cavities at the time of their operation. Furthermore, of the 33 patients who had a study of gastric or sputum cultures for drug susceptibility only three had mycrobacteria which were susceptible to all three standard antituberculosis drugs. Twelve of the patients were partially resistant to at least two of the three major drugs and 18 patients were completely resistant to all drugs. It may also be assumed that most, if not all, of the remaining 25 patients with open positive disease of considerable duration were also drug resistant.

This group of 71 patients had 73 operations. There were eight pneumonectomies, 57 lobectomies or lobectomy plus wedge excision and/or segment and only eight primary segmental resections. In most instances, lobectomy was considered the procedure of choice for patients who were open

positive with proven or suspected drug resistance. These operations were all performed through standard posterolateral incisions, and standard techniques for excision of pulmonary tissue were employed. A pre-resection space reducing thoracoplasty was performed in one patient who had extensive disease in whom it was anticipated that space problem might ensue. Five patients had reduction of the space by prior thoracoplasty which had failed to control their disease. In two instances concomitant space reducing thoracoplasty was carried out. Post-resection space reducing chest wall procedures were performed in five additional patients to obliterate persisting air spaces or to further control disease.

Results

The results of our surgical program in this group of 71 patients are also summarized in table II. In accord with current practice, patients have been grouped according to their bacteriological situation within three months of the time of operation, as well as to the presence or absence of cavity. Thus, the patients are listed as open positive or negative, or closed positive or negative.

Of nine patients who were open negative cases, results were as follows:

- Seven (7) entirely satisfactory radiological and bacteriological results.
- One (1) became positive approximately six months following lobectomy and remained so until his death from far advanced tuberculosis, death occurring two years following his resection.
- One (1) developed a space problem and a positive sputum requiring further surgery (performed elsewhere) and is now classified as inactive.

Of three patients who were closed negative (by laminograms), results were as follows:

- Two (2) patients with large necrotic residuals have had entirely satisfactory results.
- One (1) patient who had tuberculous bronchiectasis with sporadically positive sputum has also obtained satisfactory result.

Of 59 patients who were open positive at the time of operation, results were as follows:

- Forty-one (41) immediately converted bacteriologically as regards sputum and gastric cultures and have achieved satisfactory results both clinically and radiographically.
- One (1) scheduled for bilateral operation was positive after the first stage and the second stage has been performed too recently for culturing of the sputum, but everything points toward very good result.
- Five (5) single positive sputum or gastric cultures for tuberculosis following operation and all subsequent cultures have been consistently negative. These patients are also considered to have had satisfactory results. This supports the belief that an occasional positive culture following operation does not mitigate against an entirely satisfactory result.
- Five (5) failed to achieve sputum conversion following operation. Three of these had demonstrable or probable small bronchopleural fistulas. One of these was a patient who had concomitant thoracoplasty. He currently is receiving further therapy. A second patient developed a persistent space problem with a small bronchopleural fistula and has also remained positive. This patient has refused further space reducing thoracoplasty. Two patients have recently had further surgical treatment (thoracoplasty) to control disease on the operated side. One of these patients had silico-tuberculosis of the right upper lobe and remains positive.
- One (1) scheduled for bilateral operation, was positive after the first stage and the second stage has not yet been performed.
- Six (6) deaths in this group of 59 patients. One of these was due to cardiac arrest due to anesthetic complication. One patient died approximately one week following operation due to atelectasis and pulmonary insufficiency. A third patient a man of 60 with exten-

sive emphysema and marginal pulmonary reserve, died one month following operation due to pulmonary insufficiency. Three other patients died of bronchopleural fistula with spread of disease one month, three months and five months following operation. Failure in these instances possibly was a result of poor case selection in the presence of drug resistance and or due to failure to perform a prior space reducing procedure before undertaking pulmonary resection.

Discussion

A number of factors affect the results of pulmonary resections for tuberculosis. Among these are the extent of the disease, age of the patients, presence or absence of open cavities, and the presence or absence of drug resistant organisms. The first of these two factors may be controlled by rigid selection of patients for operation. Andrews and associates found that of 48 patients with far advanced disease, 27 per cent had major complications compared with 13 per cent in the moderately advanced. Wilson had also suggested the importance of the extent and severity of the early disease process as perhaps more important factors than drug resistance in surgical mortality. However, exclusion of older patients from surgical treatment and inclusion of only good risk patients with minimal or moderate disease denies hope to many patients possible of salvage at a moderate increase in morbidity and mortality. There obviously can be no hard and fixed method for screening marginal patients. Where there seemed any reasonable chance of helping our patients, excepting the aged "good chronics" or those with poor pulmonary reserve or other serious medical conditions, we have recommended surgical treatment.

The last two factors relate to the timing of operation from the initiation of medical treatment. Despite the number and effectiveness of drugs available today for the treatment of pulmonary tuberculosis, it is mandatory to avoid a continuation of medical therapy indefinitely. Surgical treatment should be recommended when the exudative disease is stabilized and particularly when open cavitary disease is evident after an initial treatment program. Holland, et al., in presenting a group of patients with cavitary disease have shown that after six months of drug therapy 29 per cent still had residual cavities and positive sputum, and after 12 months, 18 per cent had positive sputum and persistent cavities. Most authorities agree that the "target point" for consideration of operation is within six to nine months of the original treatment of the disease. Furthermore, patients who are retreatment cases or those who present multiple cavities or cavities larger than 3 cm. upon initial admission films, are particularly prone to relapse and should be always considered candidates for surgical treatment.

Drug resistance plays a prominent role in the increase of morbidity and mortality. Sweetman and Salyer found a complication role of 34.8 per cent in their resistant group as compared to 7.2 per cent in their total series of 405 patients. Moreover, the mortality was 10.9 per cent in their resistant cases as compared with 1.5 per cent in patients without such evidence of resistance. Holland and his associates have achieved an inactive state in 95 per cent of drug susceptible groups of patients but only 71 per cent in the patients with resistant organisms.

Raleigh and Steele summarized the Veterans Administration experience in over 5000 patients who had resections between 1952 and 1956. There was a 10 per cent mortality rate and a 20 per cent incidence of empyema for pneumonectomy and 11 per cent and 3.5 per cent rate respecively for lobectomy in those patients positive prior to operation. The mortality rate almost tripled and the morbidity rate was four times as great in lobectomies in patients with positive sputum as contrasted to those with negative sputum prior to operation. Murphy reported satisfactory results in 77.6 per cent where operations were performed for cavitary disease as compared to 94.7 per cent where closed lesions were resected. In resections where the sputum was positive, 82.8 per cent of the patients had satisfactory results as compared to 94.4 per cent where it was negative.

It is well recognized, therefore, that the

presence of open cavities and drug resistance can be expected to lead to higher failure rate in any surgical experience.

Of the 59 patients in this series who had open positive disease, good results with sputum conversion and satisfactory radiological appearance has been achieved in at least 47, or 80 per cent. This experience compares favorably with that achieved in similar groups of cases reported in the literature. It is also evident that open positiveness and drug-resistance are not a contraindication to operation since over 80 per cent of these patients have had excellent results with restoration to their useful place in Oklahoma society.

Summary

An analysis of the surgical resection program at the Eastern Oklahoma Tuberculosis Sanatorium initiated in April, 1955, has been analyzed through April, 1959. During this time, 73 resections have been performed on 71 patients.

Of the 71 patients, nine had open negative disease. Good results were obtained in all but one of these cases.

Three patients had operations performed for large and necrotic residues or extensive nodular disease with good results. A satisfactory result was also obtained in one patient who was operated for tuberculosis bronchiestasis.

Of 59 open positive cases, excellent results were achieved in 80 per cent. Three of these patients are considered as treatment failures. There were six deaths in this group, one of which was due to an anesthetic complication. Two patients succumbed within 60 days of the operation and three more expired later post-operatively.

The results of this study indicate the satisfactory results from surgical treatment that may be obtained in a small sanatorium. It further supports continuation of an active and progressive surgical program.

John M. Carey, M.D.
430 N.W. 12th Street, Oklahoma City, Oklahoma

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A Confirmed Hospital Epidemic of Asian Influenza*

HAROLD G. MUCHMORE, M.D.
FRANCES G. FELTON, Ph.D.
L. VERNON SCOTT, Sc.D.

A specialist in Internal Medicine, Harold G. Muchmore, M.D., graduated from the University of Oklahoma School of Medicine in 1946, where he is now Assistant Professor of Medicine.

Frances G. Felton, Ph.D., graduated from the University of Oklahoma School of Medicine in 1958, where she is now Assistant Professor of Research Microbiology.

Doctor Felton is a member of the Society of American Bacteriologists, the Oklahoma Academy of Science, the Sigma Xi and the American Trudeau Society.

L. Vernon Scott, Sc. D., received his Master of Science degree from the University of Oklahoma in 1947 and his Doctor of Science degree from Johns Hopkins University in 1950. His specialty is Virology. At the present time Doctor Scott is Associate Professor of Microbiology, at the University of Oklahoma School of Medicine.

Doctor Scott is a member of the Society of American Bacteriologists, the American Association for Advancement of Science, and Associate Member of the Society of Experimental Biology and Medicine, the Oklahoma Academy of Science, the New York Academy of Science, and the Sigma Xi.

Introduction

IN 1957 a new strain of type A influenza virus was isolated from patients in Hong Kong and Japan, and was described by Meyer, et al.[1] The disease produced by this virus is typical influenza and is characterized by headache, fever, chilliness and generalized aching. The disease has been popularly called "Asian Flu." Widespread epidemics occurred in the United States and throughout the world in late 1957 and early 1958.[2] Incidence of Asian "flu" decreased during the fall and winter of 1958-1959, but a widespread epidemic which centered in Los Angeles, California was reported in December, 1959 by the United States Public Health Service. The present paper describes a limited, but well confirmed, outbreak caused by this virus which occurred at the Veterans Hospital in Oklahoma City, Oklahoma in January, 1960.

Description of Outbreak

The incidence of febrile illnesses in patients and employees at the 488 bed Veterans Hospital in Oklahoma City, Oklahoma was about the same as during previous winters until January 20, 1960. On the afternoon of that date several patients on the neurological ward began to complain of headache, chilly sensations and aching and by midnight 11 of the 39 patients on this ward had temperatures of 100° F. or higher. During the next 24 hours a total of 16 patients showed fever over 100° F. In all, 19 patients showed fever during the outbreak. An additional number complained of similar symptoms, but had no fever. Vomiting and diarrhea were quite infrequent. The course of the epidemic as indicated by fever is shown in figure 1. Influenza was considered the most likely cause of the outbreak, and admissions to the ward and inter-ward transfers were immediately discontinued and several afebrile patients were discharged from the hospital to decrease crowding on the ward. On January 22, 1960, throat washings from five patients whose symptoms were considered typical were obtained in sterile nutrient broth and submitted to the Oklahoma State Health Department Laboratory for virus isolation. Acute phase serum was obtained on this date. Convalescent phase serum specimens were obtained February 2, 1960, 11 days later. The outbreak subsided quickly.

On January 25, 1960, the attending physician and two of three resident physicians as-

*From the Departments of Medicine and Microbiology, University of Oklahoma School of Medicine and Medical Service, Veterans Hospital, Oklahoma City, Oklahoma.

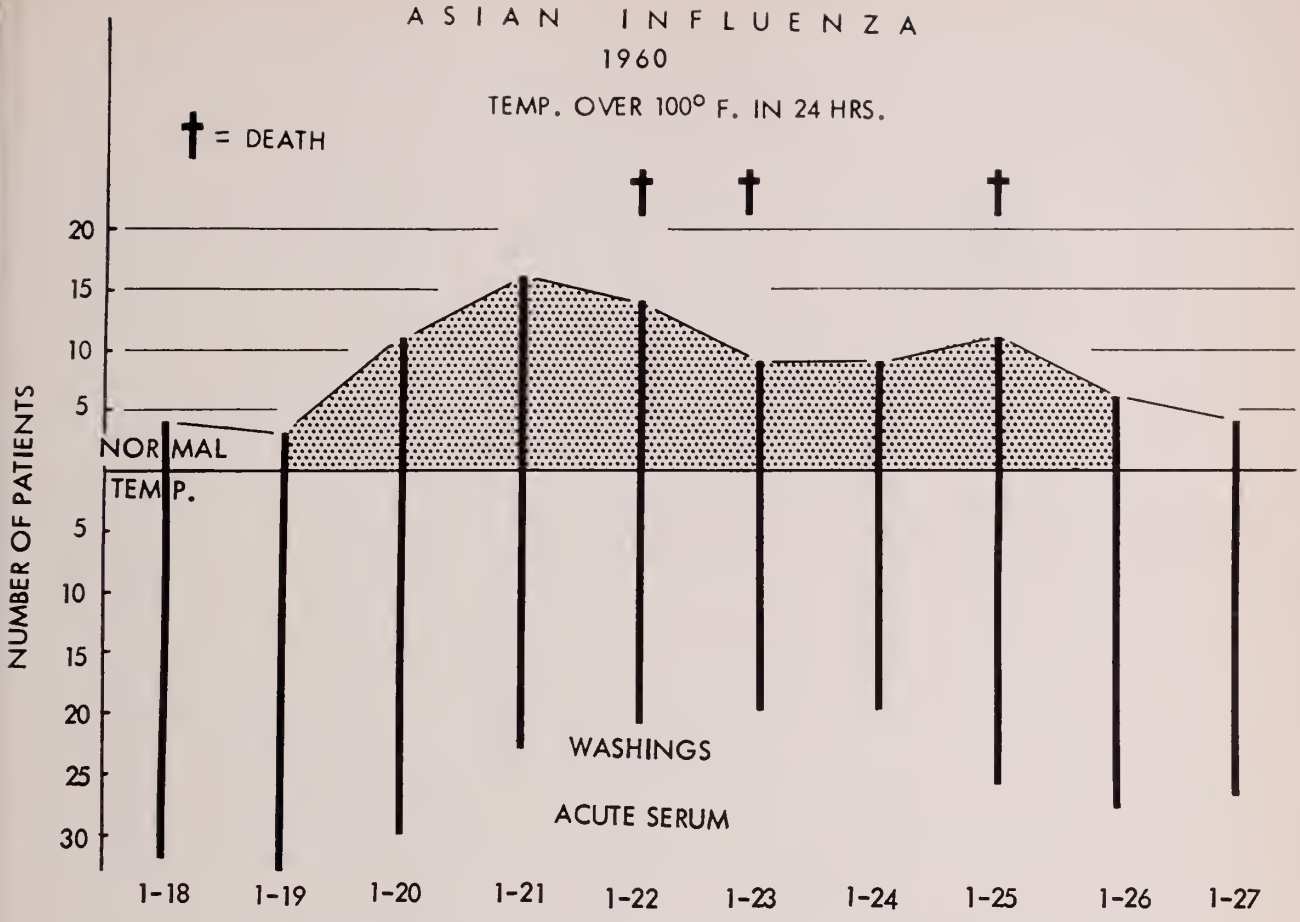


Figure 1. Each vertical bar indicates the number of patients who had fever (oral temperature over 100° F. or higher) and those with normal temperature during each 24 hour period. The stippled area represents the observed epidemic.

signed to this ward were incapacitated with influenza-like symptoms which followed a characteristic course. None of the nursing personnel assigned on this ward exhibited definite symptoms suggesting this disease. As indicated in figure 1, three deaths occurred. These three patients were chronically ill and debilitated, one suffering from Guillain-Barre syndrome, another with myasthenia gravis and a third with traumatic quadriplegia with a tracheostomy. At the time of death each of these patients was febrile, with symptoms of upper respiratory disease and aching. At autopsy on two of these fatalities acute pneumonitis was observed and microscopic sections of bronchial mucosa revealed loss of epithelium and submucosal inflammation compatible with recent influenza virus infection. Unfortunately, no specimens for viral studies were obtained from these patients.

Laboratory Confirmation

Throat washings obtained from these patients were submitted to the Laboratory Section of the Oklahoma State Health Department, for viral isolation in embryonated hen eggs. Results of isolation of virus by this method are shown in table 1.

Serum specimens obtained from these patients on the day of onset and again 11 days later were examined by the virus complement fixation (CF) test. Using this procedure the antibody titers to strains PR-8 and FM-1 (both type A influenza viruses) were determined at the laboratory of the Oklahoma State Health Department. PR-8 is a strain of type A virus isolated in 1934, and FM-1 is an A-prime strain isolated in 1947. The results of the complement fixation test are recorded in table 1. The increase in the antibody titer identifies the causal agent as a type A influenza virus, but does not indicate the specific strain.

In order to determine the strain of type A influenza involved, portions of the same

TABLE 1. RESULTS OF LABORATORY EXAMINATIONS.

INFLUENZA LABORATORY CONFIRMATION
1960

PATIENT	SERUM SPECIMEN	SEROLOGICAL TESTS						ISOLATION OF VIRUS
		HI TEST				Comp. Fix.		
		A	A1	A2	B	A	A_1	
A	1/22	128*	128	<8	64	16	8	A_2
	2/2	128	128	<8	64	64	32	
G	1/22	256	128	<8	512	64	32	NONE
	2/2	256	128	<8	512	128	64	
O	1/22	512	256	<8	256	8	4	A_2
	2/2	512	256	32	256	64	32	
S	1/22	256	128	32	256	32	32	NONE
	2/2	256	128	32	256	32	32	
T	1/22	128	128	32	128	32	16	NONE
	2/2	128	128	32	128	64	32	
Employees C	1/25	1024	256	32	512	32	16	A_2
	2/2	1024	256	128	512	64	32	
M	1/28	512	512	<8	128	16	8	A_2
	2/1	512	512	32	256	64	32	
N	1/25	512	256	64	128	16	16	A_2
	2/1	512	256	256	128	32	32	

*Titer shown is reciprocal of serum dilution.

acute and convalescent serum samples (as used in the CF test above) were examined for antibody content by the hemagglutination-inhibition (HI) test.[3] Three strains of type A influenza virus (PR-8; Cuppett, an A-prime strain; and Asian), and Lee strain of type B virus were used as antigens in this procedure. These tests were performed in the laboratories of the Oklahoma City Veterans Hospital. The results of these examinations are contained in table 1.

Asian influenza virus was isolated from the throat washings of two of the five hospitalized patients and three hospital employees. In the serologic tests (CF and HI) a four-fold increase in antibody titer between the acute phase and convalescent phase serum specimens is considered of diagnostic significance.[3] As noted in table 1 a significant rise in antibody titer to Asian virus antigen (HI test) was detected in the serum specimens of all persons from whom the virus was isolated except patient "A". There was no significant rise in HI antibody titer to any of the other viral antigens used. A four-fold rise in the CF antibody titer to type A influenza virus was noted in the convalescent phase serum specimens from patients "A" and "O" and in employee "M".

Isolation and identification of the Asian influenza virus established this etiologic agent in the patients and personnel exhibiting clinical symptoms characteristic of influenza. The results of the HI test on the acute and convalescent serum specimens further confirms that the Asian influenza virus (type A_2) was the cause of this outbreak.

Discussion

After the subsidence of the outbreak on the neurological ward, several patients on other medical and psychiatric wards exhibited fever and symptoms suggesting the influenzal syndrome. Including the patients on the neurological ward a total of 77 of 864 persons hospitalized during January, 1960 complained of upper respiratory disease associated with aching and fever. The patient attack rate for the "epidemic" ward was 19 of 39 or nearly 50%, which is a much higher rate than the approximate 9% observed for the entire hospital population.

Following the sudden outbreak on the neurological ward the experience of the entire hospital for the month of January, 1960 was reviewed. On one surgical ward five temperature elevations of 100° F. were observed in early January and some of these patients had respiratory symptoms but no laboratory specimens were obtained for viral studies. Although these incidents may be related to the epidemic no laboratory data are available to support such conjectures.

During and since this episode the incidence of illness in hospital employees has showed no difference from that seen last year at this time. However, several employees were sent off duty with acute upper respiratory symptoms. Throat washings and appropriate serum specimens were obtained from three of these. As shown in table 1, Asian influenza virus was isolated from these three employees and the infection was confirmed by a significant rise in the HI antibody titer in the convalescent serum specimen.

Recognition and identification of the causal agent of an outbreak of disease is relatively simple provided appropriate and satisfactory specimens are supplied to the diagnostic laboratory. For viral diagnosis specimens must include material obtained at the onset of the disease in individual patients. Serologic confirmation demands paired serum specimens taken at the onset and at appropriate intervals thereafter. Adequate care must be used in obtaining and preserving these specimens until they are subjected to laboratory diagnostic procedures. The Laboratory of the Oklahoma State Department of Health is equipped to perform procedures of this type. The clinical history of the disease in an individual patient is a valuable guide to the selection of appropriate laboratory procedures to be used.

No immunization procedures were attempted either on the patients on the epidemic ward or in the rest of the hospital. Since protection by vaccination probably requires four to six weeks after the initial injection,[2] this procedure was not considered feasible. In addition no vaccine whatsoever was available.

The experience reported here corroborates the observations of others that fatalities following influenza occur in debilitated and chronically ill persons, especially those with respiratory disease. It should be noted that each of the fatalities which occurred in this outbreak had respiratory problems. Thus, immunization of patients in these categories might be life-saving and should be done. However, these procedures should be initiated well in advance of the possible outbreak to afford protection. Because of the occurrence of influenza outbreaks during the winter months a vaccination program should be started in early fall of each year. To accomplish this, it is important to seek viral isolation and identification from the first suspected cases of influenza seen in a community.

Summary

A limited hospital outbreak of Asian influenza occurred in January, 1960 at the Veterans Hospital in Oklahoma City, Oklahoma. Simple epidemiological and laboratory procedures necessary for identification of the virus and confirmation of the infection are discussed. Measures to reduce crowding of patients and inter-ward traffic were apparently helpful in controlling spread of the disease.

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Harold G. Muchmore, M.D.
921 N.E. 13th, Oklahoma City, Oklahoma

MEDICAL  CENTER

Message from the Dean

Physicians throughout the nation are becoming increasingly concerned with their need for effective and continuing medical educational opportunities. Many of the problems pertaining to this field are similar to those that have confronted other phases of medical education earlier in the century.

Some of the problems include: A lack of clear definition of objectives, a wide range in the educational value of the courses and programs offered, insufficient opportunities for physicians to participate in educationally sound programs, and difficulties in financing.

Diversification of interests among modern physicians causes the production of programs to be most complex if all are to be satisfied with the postgraduate offerings.

Opportunities provided at the postgraduate level should—as a result of current advances in the basic sciences and clinical fields—favorably augment and modify an adequate initial education. Vital new discoveries become helpful only after practicing physicians learn to apply them. How best to meet this educational challenge is the broad problem of continuing medical education.

The primary mechanisms of adding to and maintaining one's professional ability include medical literature, discussions with colleagues, hospital staff meetings, local, state and national meetings, formal postgraduate courses and hospital-medical school educational programs.

One trend of postgraduate education today is toward greater emphasis on programs in which the physician is an active participant rather than a passive member of the "audience" at a conference or symposium.

Another is toward regional hospital-medical school cooperative educational efforts, which are becoming increasingly popular throughout the nation.

Teaching methods are continually being improved, as are the various teaching aids. The impact of one of the newer teaching tools—television—is yet to be realized. Its use is now under careful evaluation at our own Medical Center.

Mark R Everett

Medical Statistics: Its Concepts and Uses

EDWARD N. BRANDT, JR., M.S.

Edward N. Brandt, Jr., M.S., is a senior medical student and N.I.H. Trainee in Biostatistics in the Biostatistical Unit of the Department of Preventive Medicine and Public Health, University of Oklahoma Medical School.

Introduction

BECAUSE of the recognition of the importance or more critical and objective analyses of data, statistics is rapidly taking its place among the many disciplines that contribute to a greater knowledge of medicine. As a result, statistical terminology and procedures are appearing more and more in the medical literature, and so, if a physician is to critically evaluate the literature, he must be familiar with the concepts and uses of this young science. Although this paper can do no more than give you a glimpse of statistics, it will afford at least some insight into the concepts and uses of this tool.

Definition

The word "statistics" has been defined by Mainland[1] as "the art and science of collecting and analyzing data in such a way as to provide valid conclusions." Actually, statistics has two meanings. The first of these is as a plural noun and indicates masses of figures of which the best example is "vital statistics." The second meaning is as a singular noun indicating methods for the analysis of data. Both of these are important in the application to medicine;[2,3,4,5] however, the remainder of this paper will be primarily devoted to the second one.

Brief History

The first publication in statistics was a book entitled "Natural and Political Observations Made Upon the Bills of Mortality."[6] It was published in 1662 by Graunt, a London "haberdasher of small wares," and was the culmination of 31 years of study of the birth and death rates. Thirty years later, a paper on mortality rates was written by Edmund Halley, an English scientist for whom "Halley's Comet" was named. He subsequently published two more articles, and these three form the basis of the modern concepts of life expectancy.[7]

For the next two hundred years, there were essentially no new contributions in statistics per se; however, during this period, much of the basic work in the theory of probability was accomplished[7] by such brilliant mathematicians as LaPlace,[8] Bernoulli, Poisson, Tchebycheff, Markoff, and Nicole.

The first fruitful period in the formal development of statistics was in the early

1900's, and was primarily due to an Irish brewer named Gossett. He wrote under the pseudonym of "Student," and is best known for the development of a statistical test called "Student's t test" although he made many other important contributions.

Many significant advances in statistics have been due to Sir Ronald A. Fisher who is known as the "father of statistics." Sir Fisher has not only made many original contributions to statistical theory and methodology, but was also the first person to make the accumulated statistical knowledge available to non-statisticians by means of his two well-known books.[9,10]

The greatest developments in statistics have occurred in the past 30 years, and we now have many powerful tools available including decision theory,[11] sequential analysis,[12,13] high-speed computers,[14] non-parametric methods,[15] and others all of which are contributing to our understanding of both old and new medical problems.[4,5,16-20]

Foundations of Statistics

Statistics, like all experimental sciences, is inductive. Statistics cannot prove anything absolutely. It can only give the probability of the occurrence of the experimental results if chance alone were operating.[21] For example, let us consider the following hypothetical experiment.[21] Assume an experimenter were interested in the therapeutic effects of streptomycin as opposed to bed rest in the treatment of active pulmonary tuberculosis, and furthermore, assume that he had 107 tuberculous patients of which 55 were treated with streptomycin with four deaths, and 52 were treated with bed rest with 14 deaths. Following analysis, the statistician reported that streptomycin is apparently the better therapy since the above results would occur less than one time in 50 if chance alone were operating. What he is saying is that if one were to take 107 cards of which 18 were labeled "death" and then randomly toss 55 of these cards into a box labeled "streptomycin" and 52 into a box labeled "bed rest," on the average, one would get 14 "death" cards in the "bed rest" box and four "death" cards in the "streptomycin" box less than one time in 50 tosses. Statisticians use formulae which will give an accurate prediction of these same results without actually tossing the 107 cards. These formulae are developed by "mathematical statisticians."[23]

One must remember that the information contained in any set of data is fixed and that no statistical manipulations can create information that is not present.[22] Therefore, no statistical analysis is more or less valid than the data upon which it is performed.

General Concept

The over-all goal of statistics is to describe the properties of a general population by analyzing a small but representative portion of that population called a sample. To accomplish this, various procedures have been developed. These are based on two general assumptions concerning the data, namely that there is minimum variation among factors which are not being tested and that bias, which is any effect making a sample different from what it purports to be, has been eliminated.[24] To fulfill these assumptions, the statistician adheres to three principles[10] including: randomization which is the process of selecting patients such that each patient in the population has an equal chance of selection; replication, i.e., selecting a sample of adequate size; and control, that is maintaining the effects of extraneous factors at a minimum. Randomization results in the elimination of bias, replication results in minimum variation among extraneous factors, and control accomplishes both of these goals.

For example, let us consider some of the major sources of variation[3] in a clinical laboratory determination such as the BSP retention test.[20] These include the desired variation from patient to patient, but also there is the variation between specimens from the same patient, day to day variation for any one specimen, and variation in the assay technique itself. All of these must be considered in the analysis of the effectiveness of any diagnostic test.[16]

Design of Experiments

This brings us to what I consider the most important contribution of a statistician, namely the designing of experiments such

that meaningful and valid conclusions may be obtained from the data. Any experiment that collects data for statistical evaluation should be designed by or with a statistician in order that the following four items[3] may be included in the design. (1) Exact formulation of the experimental problem and a guide to answering subsequent questions; (2) unbiased data collection via proper control and randomization; (3) saving on resources by designing the analysis such that the questions can be answered with a minimum amount of data; and (4) proper significance testing by satisfying all of the assumptions of the desired test.[4,24]

In designing an experiment, a statistician demands specific statements from the experimenter in three major areas.[25] These statements are: (1) explicit hypothesization, i.e., exactly what is the question to be answered by the experiment; (2) model formulation, i.e., what are the practical problems as to available subjects, money, etc., and are the results of the experiment to have a general application or a narrow one (that is, are the results to be limited to the experimental subjects themselves as in a case report) ; and (3) significance level selection, i.e., with what probability do you want to state your conclusions? In medicine, 5% is the traditional level, but perhaps 1% might be better in an individual experiment.

Basic Terminology

The first term to be discussed is a simple but quite important quantity called the arithmetic mean or simply the mean. It is the quotient obtained by dividing the sum of a group of quantities by the number of quantities in the group.[26] The difference between the mean and each of the quantities contributing to the mean is called the deviation from the mean, and a short reflection will confirm the important property that the sum of deviations from the mean (retaining their positive or negative sign) is zero. This is one of the reasons that statisticians concern themselves with the square of the deviations since all squared quantities are positive numbers (or zero), and therefore, the sum of the squares of deviations will be a positive number (or zero).

In addition to the mean, one is also interested in some measure of the variation of the data from the mean.[26] The measure which is usually used is called the variance and it is an estimate of the average of the squared deviations from the mean. Another measure of the variation of the data is the standard deviation which is the square root of the variance, i.e., the square root of the average squared deviation from the mean. Intuitively, one can visualize these quantities as giving a relative idea of the amount of variation or "scatter" present in a set of data. That is, if two sets of data have the same mean but different standard deviations, one might tentatively assume that these two samples are from different general populations, and there are specific procedures for the testing of this hypothesis.[23]

The most misunderstood of all statistical statements is that "these results are statistically significant." In the first place, statistical significance has meaning only with respect to a specific probability. In other words, a correct statement would be "these results are statistically significant with a probability of 5%" (symbolically $p<0.05$), which means that the results would occur less than five times out of 100 similar experiments if chance alone were operating. What is often overlooked is that "statistical significance" has meaning only in light of the experimental situation, and that the analysis is not complete until statements are made concerning what these results mean with regard to the process under investigation. For this reason, results which are not "statistically significant" are often very important and should be published for it is just as important to know that drug A is not effective as it is to know that drug B is effective. This concept is often overlooked by experimenters and medical journal editors who are interested only in "significant" results.

Common Tests

The five most commonly used statistical procedures are the Chi Square analysis, Student's t test, analysis of variance, regression analysis, and correlation analysis. In each of these tests, one performs[24] certain arithmetical manipulations and ultimately arrives

at a numerical value. This number is then compared to a table of values which have been obtained from the solution of rather elaborate mathematical equations. The comparison of the computed value with the tabulated value gives the probability of the occurrence of such a value, and therefore, the "significance" of the results.

The chi square test was developed by Karl Pearson in 1899,[7] and its primary use in medicine is as a comparison of incidences. For example,[27] recent articles have implicated a relationship between the ABO blood groups and peptic ulceration. Data thus far collected indicates that the incidence of peptic ulcer patients with blood group O is higher than in control groups as compared by the chi square test. In this procedure, one assumes that the incidence of group O is the same in the peptic ulcer patients as in the control group. Then using the control data, one predicts the number of peptic ulcer patients that would be of the various blood types if the assumption were true. The deviation between the predicted and the actual numbers is then squared and divided by the predicted value, and this final quantity is compared to tabulated values to obtain the probability of its occurrence.

Since the means of sets of data are so easy to obtain, W. H. Gossett developed a test whereby the means of data from two different samples may be compared as to whether or not they are significantly different. For example, if one were interested in whether two drugs differed in their mean effects, the "t test" could be considered. However, this test which is relatively simple to perform is also much abused because of the nature of the restrictions on the data which may be compared. That is, the two sets of data to be compared must be randomly selected, be from normally distributed populations, and their parent populations must have the same population variance. This latter requirement, which is an extremely important one, is often overlooked and many unwarranted conclusions are drawn from improper application of this procedure to data not satisfying the assumptions.

As mentioned earlier, many populations can be at least partially characterized by knowledge of their means and variances. Therefore, one method for the comparison of sets of data as to whether or not they differ, i.e., come from different populations, is to compare their variances. In order to handle this problem, Sir R. A. Fisher developed a procedure known as the analysis of variance which in its simplest form is the ratio of the variance betwen the sets of data to the variance within the sets. This ratio is known as the "F" ratio,[24] and the calculated value is compared with tabulated values. When only two groups of data are compared, this ratio is mathematically related to both the "t" and chi square values, and if one of these three values is known, then the other two can be obtained. Therefore, any argument that one of these three tests is "better" than the others is meaningless. In this situation then the reasons for choosing one of these procedures over the others are for computational simplicity and adherence to the requirements placed on the data.

Regression analysis is the process by which an algebraic equation relating two quantities is obtained. For example, one might find the equation relating the urine volume to the dose of diuretic administered. Here one would have an equation of the form: urine volume $= a + b \times$ diuretic dose $+ c \times$ (diuretic dose)^2 where a, b, and c are constants obtained by the analysis. Correlation analysis on the other hand is a numerical evaluation of the relationship between two measurements on the same individuals. By the appropriate manipulations of these two measurements, one arrives at a figure known as the coefficient of correlation which is symbolically expressed by "r". This has a maximum value of $+1$ and a minimum value of -1. A value of $r = 0$ means that there is no relationship between the two measurements, $r = +1$ means that the two measurements are directly related, and $r = -1$ means that they are inversely related. Again, the r value is compared with appropriate tables to determine the probability of its occurrence due to chance alone. As one can readily see from this discussion, correlation and regression analyses are intimately related and in the older literature, no distinction is made between them. The commonly used forms of correlation and regres-

sion analysis are based on the assumption that a linear (i.e., straight line) relationship exists among the variables under consideration. Failure to recognize this, and applying this form of analysis to non-linear data may lead to erroneous interpretations.

Mistakes in the Use of Statistics

As reported by numerous authors,[3,6,28] the most common errors in the use of statistics in clinical medicine are eight in number, and are as follows: (1) Lack of adequate controls. This is probably the most common error and it is often very subtle. For example, are the same diagnostic criteria used on all patients, have all of the important factors been considered, etc? (2) Post hoc reasoning, i.e., misinterpretation of the data. One of the classic examples of this occurred in the 1940's when isoniazid was given to a number of patients with tuberculosis and they were soon "dancing in the halls." It was immediately assumed that isoniazid was a "miracle cure" for tuberculosis. It was several years before it was learned that this drug is also an "anti-depressive" agent and that this was the effect that had been observed rather than cure of their disease. (3) Assuming a cause and effect relationship only on the basis of a "significant" correlation. A good example of this is found in examining male patients with chronic pulmonary emphysema. In these patients, there is an extremely high incidence of benign prostatic hypertrophy. That is, there is a "significant" correlation between these two diseases, but it is unlikely that there is a cause and effect relationship between them. The high correlation is a spurious one in that it is due to the fact that both of these diseases occur primarily in the older age groups. (4) The use of "fixed totals." This occurs when one assumes that a rise or fall in percentages implies a corresponding rise or fall in absolute values. This is often not true. (5) The use of misleading averages. As an example of this, let us use a hypothetical example in which ¼ of a group of patients is treated with 100 mg. of a new drug, ¼ with 75 mg., ¼ with 25 mg., and ¼ with no drug. Then the average dose is 50 mg., but no patient actually received 50 mg. and so any conclusions based on this "average dose" are invalid. (6) The use of biased samples. One must always be sure that the sample he has drawn is an accurate representation of the population under study. The most publicized episode of this error occurred in the predictions of the outcome of the 1936 presidential election by the "Literary Digest." They predicted the defeat of Roosevelt on the basis of a sample drawn from telephone directories and automobile registration lists. Obviously this sample excludes all voters who cannot afford either a telephone or an automobile, and in 1936, this was apparently a substantial number of the eligible voters. The magazine predicted 40.9% of the total vote for Roosevelt who actually compiled 60.7% of the total vote. (7) The improper use of percentages. This is usually due to the selection of non-comparable bases from which the percentages are calculated. There are many factors making bases non-comparable, but one of these is the inability to separate significant factors. For example, suppose an experimenter reports that at his hospital over the last five years, he and his surgical colleagues had found that 54% of the patients treated for peptic ulcer with a subtotal gastrectomy and a Bilroth I gastrojejeunostomy had survived the procedure and had reported only minimal side effects while only 33% of those treated with the Bilroth II procedure had fallen into this group. In this case, any comparison of these percentages is meaningless for the effects of the different surgeons, the different stages of severity of the different patient's diseases, and the different surgical procedures are not separated and individually compared. In many cases it is impossible to separate these factors and then we say that these factors are "confounded." (8) The creation of an artificial situation in order to obtain a "statistically valid experiment." Obviously, if one is interested in comparing the effectiveness of specific therapeutic regimens in a certain disease, but in performing the experiments, certain factors important in the evaluation of the agents are rigidly controlled, then any conclusions will be meaningless unless these same controls are enforced.

In the above considerations, I have only discussed non-deliberate errors. It is obvious that one could show some improve-

ment with many forms of therapy if all patients who failed to respond were not included in the final analysis of the therapy, or if important effects in the response were deliberately overlooked.

Papers in Which Statistics Are Employed

To interpret a paper in which all conclusions are based on statistical procedures, it would be ideal if every reader were intimately familiar with all of the statistical terms and tests employed by the author. However, a person with only a limited knowledge of this subject can evaluate these papers intelligently if he will ask himself the following nine questions: (1) Who is the investigator? Is he well known and respected in his field? (2) What material has he used in the experiment? That is, what comprises the sample? Is it a fair and adequate reflection of the population with which he is working? (3) Why is he performing the experiment, i.e., what are the questions he is attempting to answer? (4) Where was the experiment performed, i.e., is the location and/or environment creating an artificial situation? (5) When was the experiment performed? This may be important if the disease studied has a seasonal variation, etc. (6) How was the experiment performed? Were proper controls taken? Are his subjects randomly selected? (7) What is being measured? Is this measurement adequate to answer his questions, and is it being done properly? (8) How many subjects is he using? Is this number adequate? (9) Has he committed one of the eight errors discussed above?

Summary

Statistical techniques are powerful but relatively new tools which when properly used contribute enormously to medical knowledge. The advent of the controlled clinical trial within the past several decades and the rich contribution to medical knowledge resulting therefrom is perhaps the best current example that this is true. A great need exists today for further adaptation of the currently available statistical methods to the problems of medical research.

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800 N.E. 13th, Oklahoma City, Oklahoma

Enteroliths as a cause of intestinal obstruction have been infrequently described in the literature. A case of obstruction of the distal colon due to this cause is presented and the literature is briefly reviewed.

Enteroliths as a Cause of Intestinal Obstruction

A Case Report

JOHN L. RITAN, M.D.

IRA ROSS GRIMES, M.D.

John L. Ritan, M.D., graduated from the University of Oklahoma School of Medicine in 1957, where he is now serving a residency in radiology.

Ira Ross Grimes, M.D., graduated from the University of Oklahoma School of Medicine in 1954 where he is now a senior resident in general surgery.

ENTEROLITHS as a cause of intestinal obstruction have been infrequently described in the literature. Graham,[1] in 1909, described a patient with acute intestinal obstruction who proved to have an enterolith in the jejunum. The nidus was a gallstone. Phillips,[2] in 1921, described two similar cases; one of the enteroliths was located in the lower ileum and the other in the mid-jejunum. In the case of the lesion in the lower ileum, a plum stone appeared to be the nidus. In 1936, Benson and Bargen[3] described an enterolith obstructing the descending colon. This lesion also had a gallstone nucleus. In 1940, Shaw[4] described obstruction of the ileum caused by an enterolith. The configuration suggested that it had originated in a small bowel diverticulum. Armitage,[5] in 1950, described a similar foreign body in the distal ileum. A fruit skin was found in the center. On a gastrointestinal examination of this patient, there were numerous diverticula of the small bowel. Finally, in 1957, Klein[6] described a large "orange size" mass in the sigmoid. This proved to be an enterolith which was diagnosed clinically. The patient was treated with Dioctyl Sodium Sulfosuccinate (Doxinate). The symptoms cleared in seven days and x-rays were normal two days later.

Case Report

The following is a case summary from the University of Oklahoma Hospital's Surgical Service:

A 53 year old female was admitted with a four to five year history of occasional bouts of sharp, knife-like pains in the left lower quadrant. Twenty-four hours prior to admission a similar attack began and became progressively more intense. Obstipation ensued and the patient subsequently passed on a small amount of flatus. The past history revealed that two years previously the patient had eaten a wild duck which had been killed with a shotgun.

Physical Examination: Blood pressure 105/70, pulse 76/min., respirations 18/min. The patient was well developed and well nourished. There were fine, moist rales in left lung base. The abdomen was slightly protuberant with generalized tenderness, more pronounced in the left lower quadrant. Rebound tenderness was present in the left lower quadrant. Bowel sounds were hypo-



Figure 1. Routine anterior posterior recumbent view of the abdomen reveals moderate gaseous distention of the colon proximal to the junction of the descendens and sigmoid. At this point there is an irregularly rounded density, about 5 cm. in diameter; somewhat uneven in texture but generally of the density of soft tissue; with a small, rounded object of metallic density near its center.

active. Sigmoidoscopy up to 25 cm. revealed a collapsed, empty sigmoid.

Laboratory Examination: Hemoglobin 14.5 gms.%, hematocrit 44%, white blood count 8,800 per cu. mm. with 86% neutrophils, 13% lymphocytes, 1% monocyte, and 1% basophil.

X-Rays: On routine x-ray examination of the abdomen, there was moderate gaseous distention proximal to the junction of the descending and sigmoid colon. At this point there was an irregularly-rounded density about 5 cm. in diameter; somewhat uneven in texture but generally of the density of soft tissue or feces; with a small, rounded object of metal density near its center (figure 1). On barium enema, the colon appeared normal up to the level of the rounded density. Very little barium flowed past this point.

A diagnosis of mechanical obstruction of the distal colon due to foreign body was made and the patient was prepared for operation.

Upon surgical exploration, the large bowel was found to be rather markedly dilated with the cecum measuring 8-10 cms. in diameter. At the junction of the descending colon and the sigmoid, the point of obstruction was apparent. The sigmoid colon at the point of obstruction was exteriorized and after closing the abdomen the colon was opened over the intraluminal mass. The mass was hard, and had an irregular, rounded surface. The bowel at this point was edematous and a thick, gray exudate was present on the external surface. Upon sectioning, the lesion had the appearance of impacted fecal material. A spherical lead fragment was found within the mass (figure 2). The gallbladder was felt to be normal. Post-



Figure 2. Radiograph of the object which was removed from the distal colon (after sectioning) confirms the presence of the metallic object within it.

operative course was uneventful and the colostomy was closed six months later.

Discussion

Wagensteen[7] reports that enteroliths are very infrequent in man but occur commonly in horses. The clinical features are those of intestinal obstruction due to any foreign body. True enteroliths have been found to consist of choleic acid and the alkaline mineral salts of phosphorus, calcium, magnesium, and ammonium. Multiple diverticula of the small bowel have been suggested as an etiological factor. In the presence of multiple diverticula of the small bowel, Wagensteen has suggested that the pouches be excised in a staged operation. In this particular case, it appears that the metallic buckshot found in the enterolith may have served as a nidus.

Summary

1. We have presented a case of distal colon obstruction due to an enterolith probably caused by ingestion of a buckshot.

2. A brief review of the literature is presented.

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800 N.E. 13th, Oklahoma City, Oklahoma

Regents Appoint Four New Faculty Members



HAASE

Gunter Roland Haase, M.D., former associate neurologist with the National Institute of Neurological Diseases and Blindness, Bethesda, Maryland, has been appointed associate professor of neurology in the departments of medicine and of psychiatry.

He is one of four new faculty members whose appointments were approved at recent meetings of University of Oklahoma Regents.

Doctor Haase was awarded his M.D. degree at the University of Munich, Germany, in 1949. He interned at St. Luke's Hospital in Denver and served a residency in psychiatry at the University of Colorado Medical School the following years.

After two years as a clinical associate in neurology at NINDB, he went to London as a clinical associate and fellow at Queen Square Hospital, returning to the National Institute in 1958.

His research includes histopathological studies of motor and sensory endings in normal and diseased muscle.

Schottstaedt to Head Department of PM and PH

New chairman of the Medical School's Department of Preventive Medicine and Public Health is William W. Schottstaedt, M.D., who moved up from the vice-chairmanship to succeed Kirk T. Mosley, M.D., now an associate dean.

Appointment of McWilson Warren, M.D., assistant professor, as vice-chairman of the department also was announced.

Doctor Schottstaedt received his M.D. degree at the University of California in 1948, interned and took residency training in medicine at the University of California Hospital. He was a Commonwealth fellow in psychosomatic medicine at Cornell University Medical College before joining the OU Medical School faculty in 1953. He is an associate professor in the departments of psychiatry and of medicine as well as in public health.

Appointed to part-time teaching positions are William S. Harrison, M.D., Chickasha, clinical assistant in medicine; Ray M. Balyeat, M.D., Oklahoma City, clinical assistant in ophthalmology; and John Francis Kane, M.D., of the Oklahoma City VA Hospital staff, clinical assistant in surgery.

ABSTRACTS

Experimental Atrophic Gastritis Associated With Inhibition of Parietal Cells

WILLIAM O. SMITH,* WALTER JOEL,** and STEWART WOLF.***

Tr. A. Am. Physicians, 71: 306, 1958

A previous communication from this laboratory reconfirmed that the intravenous administration of a normal human gastric juice to Heidenhain pouch dogs produced a marked inhibition in acid and water secretion in the stomachs of these animals. This phenomenon was first observed many years ago by Brunschwig and associates and later Code and his associates. The present paper deals with the unexpected finding of marked atrophic gastritis in the stomachs of a number of these dogs given repeated injections of normal human gastric juice over a period of time.

After as few as three or four injections several of the group of animals were noted to have a diminished output of hydrochloric acid even on control days. All four of the dogs in whom the injections were continued over a long period of time finally developed complete and absolute achlorhydria. This occurred with as few as seven injections over an 8 week period. The animals were then sacrificed and the stomachs were removed. Grossly, the stomach of each of these dogs showed definite atrophy. Microscopic examination of the stomachs from injected dogs showed various grades of cellular change characterized by disruption of the normal linear arrangement of the cells composing the branched glands, marked decrease in the number of parietal cells to the point of disappearance in some instances, a marked decrease in thickness of the mucosa with increased density, and a considerable degree of fibrosis.

The mechanism responsible for the induction of atrophic gastritis in the stomachs of these animals is not clear. Attempts to further elucidate this phenomenon are under way.

*Assistant Chief, Radioisotope Service, VA Hospital and Assistant Professor of Medicine.
**Pathologist, VA Hospital and Professor of Pathology.
***Professor and Head, Department of Medicine.

Serum Magnesium Clinical Disorders

WILLIAM O. SMITH* and JAMES F. HAMMARSTEN**

Southern Medical Journal, 51: 1116, 1958

Serum magnesium has been determined in a large group of normal subjects and in several clinical disorders.

Hypomagenesemia has been demonstrated in 5 of 12 patients with delirium tremens. The entire group shows a much lower value than the normals. All patients showed a striking response to magnesium therapy. It is believed that the intracellular level of magnesium is the important consideration.

Patients with uncomplicated portal cirrhosis have no significant change in serum magnesium.

Serum magnesium is slightly higher than normal in patients with diabetes mellitus requiring more than 20 units of insulin daily and without complications. No unusual values were found in the individual patients, however, and the difference in means appears to have no clinical significance.

A group of psychiatric patients receiving repeated shock doses of insulin showed a significant elevation of serum magnesium.

Patients with uremia have markedly elevated serum magnesium values. Patients with renal disease without azotemia occasionally have very low serum magnesiums and may develop symptoms of magnesium want.

Magnesium deficiency may occur in patients with large losses of body fluid and inadequate magnesium replacement and in patients with osteolytic disease with hypercalcemia and hypercalcuria.

*Assistant Chief, Radioisotope Service, VA Hospital and Assistant Professor of Medicine.
**Chief, Medical Service, VA Hospital and Associate Professor of Medicine.

Variability of Diuretic Response as Influenced by the State of the Organism: A Note on the Law Of Initial Values

D. C. MOCK, JR.,* A. A. KYRIAKOPOULOS,** M. L. CLARK,*** J. A. HAGANS,**** E. N. BRANDT,***** and S. G. WOLF.******

The American Journal of the Medical Sciences, 238 #2: 193-202, 1959

In a comparative study of the effects of diuretic agents upon persons free of cardiac and renal disease, the degree of increase in urine volume induced by various potent diuretics (as mercuhydrin and diamox and thiomerin) was found to vary widely from subject to subject despite precise control of fluid, caloric and electrolyte intake. Attempts to correlate these deviations with the differences in the weights of the subjects, fluctuations in emotional state or daily room temperature and humidity were unsuccessful. Instead, a significant inverse correlation was consistently found between the volume of urine excreted under control or "basal" conditions and that excreted in response to diuretic stimulation.

The use of data obtained in placebo tests provided a technique whereby it was possible to calculate the volume of urine to be expected following a control

period for a given subject if no diuretic were administered; and by comparing this expected volume (rather than the control day volume) to the volume observed after diuretic administration, it was possible to identify a response to an effective stimulus no matter what the basal or control level was. Thus correcting for the "initial value effect," originally described by Wilder, permitted the recognition of pharmacodynamic activity in groups where it might have otherwise been masked (i.e., groups where all or most of the subjects were high excretors on their control day), and prevented the false impression of marked diuresis in groups where all or the majority of individuals were low excretors on their control day.

*Assistant Professor of Medicine and Director, Experimental Therapeutic Unit.
**Research Fellow in Medicine.
***Assistant Professor of Medicine and Chief of Medical Service, Central State Hospital.
****Instructor in Medicine and Preventive Medicine and Public Health.
*****Medical Student Research Assistant.
******Professor and Head, Department of Medicine.

Intracellular Magnesium in Delirium Tremens and Uremia

WILLIAM O. SMITH* and JAMES F. HAMMARSTEN.**

Am. J. Medical Sciences, 237: 413, 1959

Erythrocyte magnesium concentrations have been measured in healthy adults, patients with delirium tremens and uremic patients with central nervous system depression.

The erythrocyte magnesium concentration was abnormally low in all patients with delirium tremens, whereas the plasma magnesium was abnormally low in only 58% of them.

The erythrocyte magnesium concentration was abnormally high in all patients with uremia and central nervous system depression, whereas 71% had elevated plasma values.

Intracellular fluid magnesium levels, as reflected by the erythrocyte, correlate better with clinical symptomatology than do extracellular fluid (serum) levels, particularly in the magnesium deficiency state.

*Assistant Chief, Radioisotope Service, VA Hospital and Assistant Professor of Medicine.
**Chief, Medical Service, VA Hospital and Associate Professor of Medicine.

Determination of Magnesium And Calcium in Urine

RAUL CARUBELLI,* WILLIAM O. SMITH,** and JAMES F. HAMMARSTEN.***

Clin. Chem., 5: 45, 1959

The renewed interest in magnesium alterations in human disease states has originated a search for simpler and more accurate analytic techniques for the determination of magnesium in body fluids. Several recent methods are based upon the chelating effect of ethylene diamine tetracetic acid (EDTA). A procedure for the determination of magnesium and calcium in urine based on EDTA titration has been developed in our laboratory.

It was found that direct titration of urinary calcium and magnesium was not feasible since the presence of a large amount of phosphate in the urine interferes with the murexide indicator used in the titration of calcium. This interference phenomenon was overcome by precipitation of the calcium with an access of ammonium oxalate in an acid solution of urine and adjusting the pH to 5.0. Recovery experiments, after addition of known quantities of calcium and magnesium to urine, were almost satisfactory with this method. Recoveries were accurate to within 3%.

*Biochemist, Oklahoma Medical Research Foundation.
**Assistant Chief, Radioisotope Service, VA Hospital and Assistant Professor of Medicine.
***Chief, Medical Service, VA Hospital and Associate Professor of Medicine.

Renal Tubular Secretion of Magnesium in Dogs

H. EARL GINN,* W. O. SMITH,** JAMES F. HAMMARSTEN,*** and D. D. SNYDER.****

Proc. Soc. Exper. Biol. and Med., 101: 691, 1959.

The mechanism for the renal excretion of magnesium has not been determined. Previous attempts to determine whether magnesium is secreted by the renal tubule have been unsuccessful. The development of the stop flow technique for localization of tubular transport along nephrons has made possible a new approach for the study of this problem in animals.

Stop flow studies were carried out in 6 female dogs; 6.3 to 25 microcurries of magnesium-28 were injected with the PAH and inulin markers. The serial urine samples delivered from the ureteral catheter were measured for the appearance of radioactivity as well as the usual determinations involved in the stop flow type of anaylsis. Magnesium blood levels were also obtained.

In each study the magnesium-28 appeared just proximal to the sharp peaks in pH and potassium change, an average of 8 samples prior to the appearance of PAH and 16 samples prior to the appearance of inulin. This consistent appearance of magnesium-28 in the urine distal to the excretion of inulin and even PAH strongly suggests that magnesium was being secreted at the level of the distal tubule or distal portion of the proximal tubule under these experimental conditions.

*Resident Physician.
**Assistant Chief, Radioisotope Service, VA Hospital and Assistant Professor of Medicine.
***Chief, Medical Service, VA Hospital and Associate Professor of Medicine.
****Resident Physician.

PRESIDENT'S LETTER



"Those who would give up essential liberty to purchase a little temporary safety deserve neither liberty nor safety."—Benjamin Franklin

This year is an election year. Every two years, election years, since the inception of the Social Security Act, the benefits have been increased, either by increasing the payments, or broadening the base of coverage.

We can expect action on some Forand type of legislation this year. The second session of the 86th Congress is not one likely to get out of the rut.

Action taken at this time will not be the result of a comprehensive view and study of our society. This action should come only after the White House Conference on Aging in 1961, if the necessity for action is then indicated.

I believe that we have reason for feeling that occasionally our legislators' "Welfare Values" are expressed in the following order:

1. Welfare of re-election of the individual.
2. Welfare of the party.
3. Welfare of the nation.

If the above order is reversed and the problem adequately studied, Congress will get out of the rut and the Forand Bill, or compromise legislation, will not be passed.

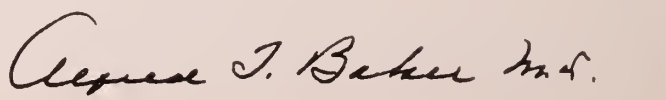
* * *

Early in December 1959, the Committee on Prepaid Medical Care mailed letters to the societies representing the twenty-nine counties with over 100% utilization of prepaid medical care plans, asking for discussion by the societies and for suggestions relative to correction.

To date of this writing, no replies have been received. This may represent voluntary inertia (plain laziness) or a willful effort to avoid a pressing problem.

If every physician would ask himself if hospitalization is essential to the treatment of the patient; and ask himself if he would insist upon hospitalization in absence of prepaid plans or Department of Public Welfare Funds; if he would not hospitalize a patient merely for his own convenience, or merely to accede to the request of a patient whose only requirement for hospitalization is the fact that they want to go to a hospital because a prepaid plan or because the Department of Public Welfare would pay the bill . . . then that portion of over-utilization due to abuse or misunderstanding of the purpose and intent of prepaid plans would be eliminated rapidly.

Such abuse is a symptom of a lack of morality that should not exist.



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It is fully expected that Aldactone will change present medical concepts of the therapeutic limitations of managing edema. Many patients living in a greater or lesser state of edematous invalidism can now be edema-free. To others, gravely ill, Aldactone will be life-saving.

When used alone, Aldactone will produce a satisfactory diuresis in about half of those patients whose edema is resistant to conventional diuretic agents.

When Aldactone is used in a comprehensive therapeutic regimen, which includes a mercurial or a thiazide diuretic, a satisfactory diuresis and relief of edema may be expected in approximately 85 per cent of edematous patients *who would not otherwise respond.*

DOSAGE: For most adult patients the optimal dosage of Aldactone, brand of spironolactone, is 100 mg. four times daily. Aldactone should be administered for at least four or five days before appraising the initial response, since the onset of therapeutic effect is gradual when it is used alone. Aldactone manifests accelerated activity with greater response as early as the first and second days when used in combination with a mercurial or thiazide diuretic.

SUPPLIED: Aldactone is supplied as compression-coated yellow tablets of 100 mg.

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Special Articles

A March to Utopia—True or Political*

ARDELL T. EVERETT

The author is a member of the Joint Committee on Social Security of the LIAA-ALC, as well as a member of several industry committees where health insurance is involved, including the Public Relations Committee on the Health Insurance Institute, member of the Board of Directors of the Health Insurance Association of America and currently the chairman of the Committee on Federal Social Problems. Also a member of the Insurance Committee of the Chamber of Commerce of the United States and chairman of the Chamber's health insurance subcommittee. In addition, he is a member of the National Advisory Committee for the 1961 White House Conference on Aging.

THE PARADE of advocates of national social insurance legislation to solve the health care problems of the aged is under way. With increased momentum through November, 1960, the parade will become a full-grown roadshow with most political aspirants using problems of the aged as a theme. While the broad subject of aging will be discussed generally, the problems of the effect of inflation on stabilized stipulated incomes will be lightly covered and medical care needs of the aged will be emphasized. Traditionally, the problems of any segment of a free society unable to assume responsibility for personal care has been assumed by the local community. To our knowledge, there is no place in the United States where health care is not available for any segment of the population needing and wanting such care.

Local Level Evaluation

Should assistance be required, the only proper evaluation of needs must be made at the local level. Any Federal health plan directed at the central national level would be cumbersome, unworkable and costly beyond comprehension.

It can be anticipated that special attention will be directed toward proposed amendments to the Social Security Act. Social Security legislation has been a popular item of consideration — and adoption — in each election year since 1950 and you can expect that 1960 will be no exception. But increased cash retirement benefits no longer loom as the only issue.

In 1958, the introduction of the Forand Bill provided the impetus to much of the current debate as to the health care needs of the aged population. The passage of a bill to call a White House Conference on Aging in January, 1961 added to the interest in the subject of aging. The wheels began to turn toward 1960 as the critical year for legislation directed at the problems of aging and the aged.

What Would It Provide?

What would Forand-type legislation do for the aged? Such bills would amend the Social Security Act to provide certain hospital, nursing home and surgical benefits to all persons eligible to receive Social Security benefits. Included as recipients, therefore, would be persons under age 65, such as widows with children under 18 and disabled beneficiaries and their dependents. *It would not cover those aged persons not eligible for Social Security benefits.* Excluded from

*Reprinted through the courtesy of The Weekly Underwriter, 116 John Street, New York 38, N.Y., issue of January 2, 1960.

benefits are the 2.5 million of the aged who may be in need as evidenced by their inclusion in Old Age Assistance rolls.

Therefore, Forand-type legislation would blanket *all* persons over age 65 still employed —but otherwise eligible for Social Security —and the retired persons covered by voluntary health insurance or otherwise able to pay for health care. Benefits are not directed to *need,* although the proponents of legislation continually cite the needy elder citizen as the reason for their advocacy.

During 1959, the parade began to form. As plans got under way for the White House Conference on Aging, the shape of things to come was unfolded. A planning institute was held for leaders of state conferences on aging. (Each state is to submit a report to the national conference based on state surveys and conferences.) Material is beginning to flow to the states to use in making surveys and planning conference programs. Included are many statistical tables which, per se, would seem to demonstrate a "need" without any suggested effort on the part of state conferees to explore the true facts of the local situation.

A publication, "Guide for State Surveys on Aging," is an example of the unanswered questions—which probably won't be asked—inherent in some of the statistics.

For one of the states, a table shows that the median income of women age 65 and over is $740 but that 63.2 per cent of them had no income. This suggests a picture of need and, indeed there probably are needy women in this group. But surely, the 63.2 per cent with no income have food, shelter and clothing furnished by the family and does not necessarily mean destitution, yet this question is unanswered by the statistical table.

Support From Husbands

Included in the number of women with no income probably are all those entirely supported by their husbands. This assumption is based on other income surveys made by the Bureau of Census in which this practice was followed. However, there is no clear presentation of the individual means of support for the group of elderly women. Unfortunately, few persons will make the effort to inquire beyond the bare figures included in such publications.

During the summer of 1959, the McNamara Committee—The Subcommittee on Problems of the Aged and Aging of the Senate Committee on Labor and Public Welfare —held public hearings in Washington. One of the key witnesses was Wilbur Cohen of the University of Michigan.

Mr. Cohen also appeared on the platform of the New Jersey Conference on Aging held in April, 1959. He feels that our economy can support tremendously improved benefits for the aged. Although the Social Security program was designed as a floor of protection on which the individual could build his retirement plans through private thrift, Mr. Cohen prefers to view it as "a floor with a kind of Bigelow carpet on it not only to keep one's feet warm but to give some aesthetic pleasure also."

Some of Cohen's proposals would revise Social Security to provide:

—hospital and nursing home care

—a 20-25 per cent increase in average benefits

—an increase in maximum taxable earnings to $9,000

—an increase in the minimum and maximum benefits

—an increase in widow's benefits from 75 per cent to 100 per cent of the primary benefit

—removal of the age 50 limitation on disability benefits

Contributed Very Little

Senator McNamara left the Washington scene this fall to hold hearings in various sections of the country. As reported in the newspapers, the hearings did little to contribute to studies in depth of the true situation of our aged population. Rather, the hearings provided a platform for individual aged persons to ask for Federal help to relieve personal problems. The emotional im-

pact of these hearings contributed little to an objective approach of studying the myriad of inter-related problems of aging.

In July, hearings once again were held by the Committee on Ways and Means of the House of Representatives. The parade continued. Fortunately, no precipitous action was taken at that time.

E. J. Faulkner, appearing in behalf of the American Life Convention, the Health Insurance Association of America and the Life Insurance Association of America, very ably presented the position of the voluntary health insurance industry. Included in his testimony was an estimate of the cost of the Forand Bill as prepared by competent actuaries in the health insurance field. First year cost estimates range from $2,074 to $2,387 million but by 1980, it is estimated that costs would be in the range of $5,981 to $7,660 million in terms of 1960 dollars. In terms of "level premium costs"—constant costs over the long range—the Forand Bill would cost from 2.32 to 2.97 per cent of taxable payroll. Since the level premium cost of present Social Security benefits is 8.76 per cent of taxable payroll, *the long term total Social Security costs would increase by at least 26 per cent to an over-all cost of 11 per cent of taxable payroll.*

Why the Opposition

In his conclusion, Mr. Faulkner stated:

"We express to you our firm opposition to HR 4700 and similar proposals for the government financing of the costs of health care because:

(1) Such measures are premised on the false assumptions that most of the aged are unable to finance their health care costs or to secure adequate voluntary health insurance.

(2) The proposal would impair, if not destroy, the present Social Security system, and voluntary health insurance.

(3) It would fail to alleviate the only real problem —that of the presently aged who require assistance to meet their health care costs.

(4) Such proposals would impose a new, uncertain, growing and crushing burden on an already heavily taxed citizenry.

(5) Such proposals would develop enormous pressures for a complete compulsory health insurance plan.

(6) Such proposals would aggravate and intensify the present and future economic and fiscal problems of the country.

(7) Such measures are unnecessary since voluntary health insurance has the capacity and the will to provide the aged, as well as the other segments of the population, with a sound and economic means of paying health care expenses.

(8) Such proposals might very well promise and impose a tax for benefits, which, based on presently available facilities, could not be delivered.

It is unnecessary for Congress to mortgage the future of Americans as a free people by enacting legislation of this type. In the best interests of us all, we ask you to vote against these proposals."

Away From Individualism

Assuming the position of Janus, our retrospective view tells us that the prospective picture for 1960 is one that clearly portrays a quickening pace in the parade away from individualism.

State conferences on aging will be held without adequate and authoritative representation of the business and professional community unless there is a stirring among that group to action. Coming as they will in the middle of election year furor, these conferences will add to the increasing publicity in the press and other communications media and will be designed to trigger emotional rather than logical, consideration of problems of aging.

It is known already that a tremendous number of bills to amend Social Security—all those envisioned by Mr. Cohen and more besides—will be introduced in Congress. The pressures generated by state conferences on aging and by political advocates may result in legislation directed to one facet of a problem thereby compounding the overall problem.

For example, can medical care problems be separated from problems of income and housing? Isn't inflation a more dire basic problem of the aged affecting ability to secure voluntary insurance, adequate diet and proper housing? It has been stated that many of the aged occupying hospital beds would be released if adequate low cost housing were available.

Little, if any, credit is given to the tremendous growth of voluntary health insurance among the aged which enables the elderly citizen to provide his own means of financing medical care.

Voluntary health insurance among the aged has grown at a more rapid pace than the growth of the number of persons living beyond age 65. It must be remembered that voluntary health insurance essentially is a product of the post-World War II period and it is only natural that the health insurance protection held by the aged population is directly related to the growth of health insurance among those under 65.

Number of Persons with Hospital Expense Protection Through Voluntary Health Insurance 1946 and 1956:

	Per Cent of Population Covered		
Year	All Ages	65-69	70 & Over
1946	30%	15%	5%
1956	69%	48%	30%

It is evident from these figures that as more health insurance is available to the working population under 65, the number covered after retirement will increase. So that within another ten years, the number of persons age 65 and over protected by voluntary health insurance should equal or exceed the percentage of the population under 65 who are covered in 1959. This comparison of the growth of voluntary health insurance is especially significant when compared to the per cent of the total population age 65 and over covered by a compulsory governmental social insurance program, namely, Social Security. At the end of 1957, after twenty years of operation of the Social Security Act, only 66.6 per cent of the aged population were eligible for Social Security retirement benefits.

Need Not Considered

Forand-type legislation would provide benefits without regard to need and, as pointed out before, would cover many who are still working or self-sufficient but would exclude those most in need. The cost could be staggering but is too often ignored politically because it would not be in the Federal budget.

The tendency has been to regard Social Security taxes as something separate and unlike other taxation which provides general funds to the federal, state, and local governments. However, it must be recognized that the Social Security tax is a part of the *total* tax load on the economy and because all governmental needs seem to be increasing, it is imperative that tax money be used efficiently in the sense that it be directed to areas of established need. By diverting tax funds to many persons who do not need governmental assistance, we reduce the capacity of our economy to provide for those aged persons who do need assistance in financing their medical care.

The voluntary health care mechanism is confronted with a real challenge to its future existence. Enactment of legislation attaching health care benefits to Social Security will be the *next to the last step toward national health insurance* for all citizens. For if health care benefits are provided to all those *eligible* for Social Security benefits, it would appear to be inevitable that such benefits be extended to those who are *paying* for them—the workers under age 65. We have only to look at the pattern of the disability benefits to discern the future for all insured and company planned employee benefit programs.

With the enactment of disability benefits at age 50, it could be foretold that those benefits would be extended to persons under 50. Once the compromise had been made, and the principle accepted for persons aged 50 and over, there is little argument that can be made against the point that a person at age 40—or 30, or 25—who is disabled is not as deserving of benefits as a person at some other age. The same approach would hold for health care benefits once the principle of adding them to Social Security *for any segment of the population* is established.

Their Real Goal

Let us not forget the ultimate goal of those who wish us to believe that the addition of health care benefits to Social Security is proposed only because of the plight of our aged population. In his testimony before the Committee on Ways and Means of the United

States House of Representatives in July, 1959, Walter P. Reuther stated: "It is no secret that the U.A.W. is officially on record as backing a program of national health insurance. But even if we were against national health insurance, we would favor the passage of the Forand Bill." Many of the prominent proponents of national social health insurance decry the fact that the United States is the only country in the world which has not enacted some type of compulsory health insurance.

They fail to note that the United States is one of the few remaining nations in the world where private enterprise may flourish and where there is any degree of individual freedom as we have known it throughout the history of this country. Could it be that there is some connection between these two points which these persons have neglected to mention?

If, through the Social Security mechanism, voluntary health insurance is obliterated from the American scene, what of the future for other forms of private insurance? For that matter, what is the future for *private enterprise* regardless of the form it may take? History tells us that the road to collectivism is entered through social "insurance" legislation. The populace is lulled by the cries of those who profess to be thinking only of the needs of the unfortunate few of the citizenry who must have help. But when the rude awakening comes, it is found that the legislation is *not* directed toward only the unfortunate few—it blankets all the citizens under a paternalistic centralized government approach. Unfortunately, by then it is too late—the pattern is irreversible.

We cannot close our eyes to social problems whether they be of the aged population or of our youth. The challenge to every citizen is whether he will shoulder his responsibility in his community to assure a comprehensive objective analysis to determine what problems exist. Once the problems, and their inter-relationship, are clearly delineated, the responsible citizen must work to arrive at the proper solution in the framework of the American climate of self-dignity, individual initiative and personal freedom.

Too often in the past few years the battles to preserve the fundamental principles which we cherish against those who espouse the philosophy of "cradle to the grave" security have been lost by default. The professionals in the field of social legislation have dominated public discussions and representatives of the business and professional groups have been notable by their absence.

Unless responsible citizens are stirred to action, the state conferences on aging will be held without authoritative representation from business and the professions. The conferences will be a hollow mockery since the format will be the stereotyped answer to all problems—Federal intervention through legislation. *Only if there is active participation by an alerted citizenry in the state and local conferences on ag ng can an objective program be developed to study and solve the problems that do exist.*

I personally believe that even beyond participation in state and local conferences on aging, each of us, regardless of party, must begin to take a more active interest in politics. Over the years, the word "politics" has acquired some unsavory connotations to many people, but remember that political participation is the very heart of our system of government. Action at the local political level—in the party of your choice—can have far reaching effects, through the county and state organizations, which eventually will be felt in Washington. Only through active expression of interest can we keep our representatives in government informed as to the real desires of their constituents.

I am convinced that most citizens want sound government and balanced budgets but unless their influence is felt by participation, their voices will not be heard. Being a good citizen means acceptance of the responsibility of making our political system work and only *you* can assure its success.

The practice of medicine may regress in the next ten years as much as it has progressed in the last decade! This article, reprinted from the AMA's publication, "PR Doctor," February 1960, tells what organized medicine must do in counter-revolution.

THE SIXTIES . . . Decade of Decision

THERE'S A REVOLUTION going on in your own backyard—a revolution that within ten years could alter your whole life, determine how you practice medicine and dictate the way you care for your patients.

The crisis will come in the '60s, perhaps within a matter of months. The decision to be made is this: who will take the leadership in solving medical and health problems?

This is no case of the "good guys" vs. the "bad guys." It is a case of Americans looking for the best way to provide medical care for everyone. The goals on each "side" are the same—the mechanisms for achieving them differ.

The whole revolution stems from a growing feeling that suitable medical care is not only important, it is a right to which everyone, regardless of his financial status, is entitled.

No doctor of medicine would quibble with the objective. Traditionally individual MDs and medical organizations have labored to care for the indigent as well as the paying patient.

The doctor, however, disagrees that a "right" implies a government handout in all cases. He would question the growing hue and cry that problems related to medical care are so acute that the government must step in to solve them. In the first place, he would like some evidence to show that these problems really exist since he hasn't been able to document them in his own experience. In the second place, he knows the government is less qualified than the medical profession to tackle the job. Above all, he bitterly resents the fact that politicians use medical care as a stepping stone to political success.

Yet people are increasingly willing to let the government assume the responsibility for health affairs. For though they bear no grudge against medical organizations, a lot of Americans feel physicians are losing this leadership by default.

This is mighty strong talk but this is a mighty serious issue.

Well, what happened to medicine? One cogent answer turns up time after time in a recent survey conducted for AMA by an independent firm. Doctors—individually and collectively are guilty of a communications failure.

The survey points up communications failures in these vital areas:

- Failure to communicate to patients understandingly and understandably about their illnesses.

- Failure to communicate effectively regarding fees and medical costs as they relate to other prices today.

• Failure to communicate effectively on a personal level with patients. This leads to complaints that the doctors are cold, indifferent to people's personal problems both medical and financial; that they don't care about people's feelings, that they make people wait too long.

• Failure to communicate the story on the long-established programs medicine has launched, i.e. to train more doctors, to get these doctors into communities needing them, to police its own ranks.

• Failure to communicate its real public interest in the case of the indigent, the aged, the economically stricken.

Medicine's planned efforts to get out of the scientific cocoon and into the community to tell its story are recent, dating back less than a decade in some cases. Much progress has been made, but will it be enough to stem the tide of support for government medicine?

Part of the communications failure may be traced to doctors' commendable reticence to promote themselves and their organized projects. Many a doctor will say, "If you do a good job, people will ultimately find out about it." But "ultimately" may be too late. Politicians are wasting no time promoting unsound and undesirable schemes to "governmentalize" medicine. They know that salesmanship of a product—or a point of view—is essential today.

Though physicians and medical societies may not be well versed in the techniques of communicating in the way that a salesman or politician does, they have the winning card. The modern medical care system in America, under constant scrutiny and undergoing continual improvement by physicians and allied medical people, is the best in the world.

Unpleasant as the realities of the need for "salesmanship" may be to the scientifically oriented physician, it is essential that he stand up for the system he knows works best—and at the same time, work to assure that it continues to be the best. He can promote his point of view in the name of organized medicine, not selfishly, but on behalf of the public interest.

Today the medical profession must *adapt its techniques to the times.* You don't use a musket in the face of a machine gun barrage. MDs, medical societies and allied groups must emphasize through every available means to the public that working to improve today's medical system will provide better medical care in the long run than substituting a government system.

The *times* are today.

The *technique* is positive action coupled with better communications.

The suggested "counter-revolutionary" approach follows:

Six for the Sixties— Make Service Your Slogan

1. *Service from a personal physician.* Americans crave this. In this area some cracks appear in the mirror reflecting the doctor image. Though physicians rate higher than dentists or lawyers on ethics and dedication, MDs are more often criticized in areas of personal service. Analysis of the opinion survey shows that the physician's own attitudes and motives along with the efficiency with which he runs his office are keys to service attitudes. Consequently, every physician should be aware of his own personal responsibility not only for his personal public relations but for the public relations of the entire profession.

Needed—Increased professional educational efforts on human relations techniques, ways to communicate to people more effectively . . . more orientation courses for new members, more refresher courses for regular members, more medical society action to establish courses in medical schools on nonscientific medical subjects.

Coming—A new booklet for physicians on the human side of medicine and medical ethics practically applied.

2. *More medical service.* Over one fifth of the Americans surveyed feel there is a doctor shortage and some intimate the profession actually limits supply. About one-fourth indicate a local need for more hospital facilities. Problems appear greater in rural areas.

Needed—All-out publicity on medicine's recently stated positive policy regarding recruiting more young people into medical

careers, offering medical scholarships, expanding medical schools, and ultimately training more doctors . . . joint efforts by medical societies and medical schools working together to tell what's being done to increase the number of medical graduates . . . local studies on ways to expand facilities, set up scholarships, as recommended at AMA's Dallas Clinical Meeting . . . awareness that the public interest is *not* served when medical societies and medical schools can't get along together.

Coming—A new career guidance handbook for counsellors and an accompanying leaflet for interested teen-agers to further career recruitment efforts.

3. *Better medical service.* Lack of prompt service, properly administered, coupled with the criticism you can't always get a doctor when you need one, are at the root of much of the criticism of the medical profession, the survey says.

Needed—Renewed attention to operation of round-the-clock emergency systems, well publicized, to demonstrate medicine's desire to provide continual service . . . more educational programs to stress the importance of securing a family doctor before emergencies arise . . . repeated study of grievance committees to make sure they are functioning effectively and that people know they exist . . . continued emphasis on office efficiency to reduce service complaints related to unnecessary waits, difficulty of access to a doctor, lack of adequate time for patients.

Available — How - to - do - it literature on emergency call systems and grievance committees, suggestions on family doctor educational campaigns, materials on increasing office efficiency.

4. *Service at reasonable price.* The weakness of medical efforts to communicate the true story on medical costs and resultant lack of understanding of financial motives leads to public feelings that doctors are excessively money-minded, that they charge a lot more than expected, and that they don't try to keep charges as low as possible.

Needed—Greater widespread educational efforts on medical care costs in relation to other costs today . . . promotion of medicine's traditional guarantee to provide medical care to all, regardless of ability to pay, whether these plans are formal or informal . . . development of relative value studies as recommended by AMA's House of Delegates among societies interested in this aspect of cost . . . continued education of individual physicians on the importance of itemizing medical bills, initiating advance fee discussions, cutting costs where possible for patients . . . increased emphasis on using not abusing health insurance plans and promotion of wider insurance coverage.

Coming—A report on a recent survey of societies' efforts to provide care to the indigent and the medically indigent and how-to-do-it materials on establishing and publicizing a guaranteed care program . . . regional conferences to explain techniques of conducting relative value studies . . . leaflets for profession and public on using, not abusing health insurance.

5. *Service beyond the call of duty.* Today doctors are less often credited than are dentists or lawyers with trying to help solve community problems. Medical societies should tackle at least *one* positive community service project in 1960, whether it be a health fair, a tetanus immunization campaign, an attempt to reduce highway accidents, or a similar effort to solve a local health problem. Since Forand type legislation is imminent unless actively countered, a positive project in the area of aging should be a PR must in 1960.

Needed—Creative origination of community service projects by medical societies . . . more community participation by physicians.

Coming—New how-to-do-it leaflets on community projects, particularly for senior citizens.

6. *Service as citizens.* Regardless of their "special interests" physicians are citizens. 1960 is an election year. Consequently, doctors as private citizens should actively encourage people to vote, work in the political parties of their choice, and generally work for good government. It is as important for medicine to support sound legislation as it is to oppose unsound bills.

Factors Relating To Physician Distribution in Oklahoma 1950-1959

Part II of a Series**

ROBERT C. LOWE, M.D.*
JAMES A. HAGANS, M.D.*
PEARL FISHER, Ph.D.*

IT HAS BEEN SAID that the concentration of population in Oklahoma, along a southwest—northeast diagonal "follows the pattern of oil development." If we add to this picture an extension of concentration to the north and to the south from the midpoint of the diagonal (perhaps related to the marketing pattern of the cattle and agricultural industries), the broad ellipses of population concentration in Oklahoma is described.

This pattern also depicts the concentration of *practicing* physicians in the state. The general parallelism is to be expected. However, the degree of population per se is not the relevant feature. Increasing concentration leads to population organization and to community development, i.e., urbanization, in some degree.

Population organization and degree of urbanization appear to be the basic features most clearly related to the concentration of physicians. Pursuing this concept, and extending the analysis begun in Part I to the whole state at the county level, the counties have been sorted (table III) into four categories; (1) those demonstrating a metropolitan type of organization, (2) those demonstrating "urbanization"*** of a non-metropolitan type, (3) those demonstrating a lesser degree of population organization into towns or cities, but with a total income level above 20 million dollars for 1958****, and designated "Rural-I" and (4) a "Rural-II" category consisting of those counties presenting the minimum degree of population organization and total income for their respective areas.

*All three of the authors are with the Department of Preventive Medicine and Public Health, University of Oklahoma School of Medicine; Doctors Hagans and Fisher are also with the Biostatistical Unit of the Department of P.M. and P.H.; in addition, Doctors Lowe and Hagans serve with the Department of Medicine.

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***"Urbanization" is defined here, exclusive of metropolitan organization, by the presence in a county of one or more communities, per 500 sq. miles, each with a 1950 census of 2500 or more; and/or the presence of a city, the population of which includes 50% or more of the county's 1950 census.

****Total income calculated from per capita income published for each county for 1958 by the University of Oklahoma Bureau of Business Research and 1958 population estimates (table IV—footnote).

Table III

ORGANIZATION LEVEL AND NET CHANGE IN:

A. Practicing Medical Physicians—1950—1959

Level of Organization	No. of Counties	No. of M.D.'s—1950	No. of M.D.'s—1959	% Change	Chi Square Analysis*
Metropolitan	2	703	780	+9.9%	
"Urbanized"	21	615	579	—5.9%	
Rural—1	9	196	163	—16.8%	
Rural—II	45	367	273	—25.6%	p <.001**
Total	77	1881	1795	—4.5 %	

B. Osteopathic Physicians—1950—1959

Level of Organization	No. of Counties	No. of D.O.'s—1950	No. of D.O.'s—1959	% Change	Chi Square Analysis*
Metropolitan	2	103	166	+61.2%	
"Urbanized"	21	104	105	+0.9 %	
Rural—I	9	42	31	—26.1 %	
Rural—II	45	74	70	—5.4 %	p <.001**
Total	77	323	372	+15.1 %	

Table IV

SIZE OF COMMUNITY IN EACH ORGANIZATION LEVEL AND NET CHANGE IN:

A. Practicing Medical Physicians—1950-1959

County Level of Organization

Community Size	"Urbanized	Rural—I	Rural—II
<2500	—35.1%	—28.8%	—31.5 %
2500—6000	—26.5%	—12.0%	—22.0 %
6000—18000	—12.1%	—11.6%	—25.0 %
18000—38000	+ 8.3%		
State Average	— 5.9%	—16.8%	—25.6 %
Chi Square Analysis* (Vertical p<.01 &	>.001**	p>.05	p>.05

B. Osteopathic Physicians—1950-1959

Community Size	"Urbanized"	Rural—I	Rural—II
<2500	+23.8%	—25.0%	—11.9%
2000-6000	— 4.8%	+25.0%	+ 3.1%
6000-18000	+13.0%	—42.8%	0.0%
18000-38000	—15.4%		
State Average	+ 0.9%	—26.1%	— 5.4%
Chi Square Analysis* (Vertical)	p>.05	p>.05	p>.05

*p represents the probability that a difference as great or greater than that observed would have occurred by chance alone; when the value was .05 or less (1 in 20 or less) it was regarded as sufficiently rare as a chance occurrence to indicate a significant difference.

**indicates statistically significant difference.

Table V

PERCENT CHANGE IN PHYSICIAN/100,000 POPULATION
BETWEEN 1950 & 1959 IN EACH
LEVEL OF ORGANIZATION

Level of Organization	M.D.'s / 100,000 1950	1959*	% Change	D.O.'s / 100,000 1950	1959*	% Change
Metropolitan	121.2	104.0	—14.2 %	17.8	22.1	+24.2 %
"Urbanized"	80.9	75.3	— 6.9 %	14.1	13.6	— 3.5 %
Rural—I	75.0	70.9	— 5.5 %	16.2	13.5	—16.7 %
Rural—II	58.2	49.5	—14.9 %	12.1	12.7	+ 5.0 %
State	80.6	77.6	— 3.7 %	13.6	16.2	+19.1 %

*Population estimates are those published by the Oklahoma Committee for Population Estimates as of July 1, 1958.

The organized communities of the last three categories have been sorted by size to illustrate the subject relationships in these sub-groups.

As the level of population organization decreases and becomes more and more rural, there is a significant tendency towards a greater and greater net loss of physicians in general over the nine year period (table III—A and B). It is noted that in the metropolitan areas the per cent increase in osteopathic physicians is about seven times that of the medical physicians. The net overall change for the state reflects a loss of 4.5% in practicing M.D.'s and a gain of 15.1% in D.O.'s.

Table IV—A and B illustrates that there is no apparent relationship between community size within levels of organization and the net change in D.O.'s and practicing M.D.'s in this period, except for M.D.'s in the communities of "urbanized" counties. Here the larger communities (i.e.>18,000) showed a net gain in M.D.'s (+8.3%), while there are increasing net losses in M.D.'s as community size decreases. This contrast suggests that further study in relation to the level of urban development at the individual community level is needed.

The data in table V (using the familiar standard of No. of physicians/100,000 population) will provide a quick orientation on the effects of the shifting of our population from one level of population organization to another. Quantitatively, in regard to M.D.'s, the metropolitan areas have fared as badly as the Rural-II level, with the minimum population concentration and organization, (a decrease of 14.2 and 14.9% respectively). The population shifts reduced a gain to a loss in the former areas and reduced a loss to a lesser loss in the latter. Interestingly, the increase of osteopathic physicians outstripped the population gain in the metropolitan areas, and the minimal decrease in the most rural area was converted to a minimal increase, relative to the population shift.

Part III of this series will deal with two further questions, (1) Why does community size appear to be significant in the "urbanized" counties but not in the others? (2) What relationship exists between the age and concentration of M.D.'s and their distribution?

News

INDEMNITY PROGRAM FOR SENIOR CITIZENS APPROVED

Council and Delegates 'OK' Plan

After nearly a year of investigation and negotiation, Oklahoma's House of Delegates met on February 21 and approved a medical-hospital pre-payment program for senior citizens. The program, Blue Cross-Blue Shield "Special 60," is an indemnity plan designed as an answer to the American Medical Association's plea (1958 Mid-Winter meeting) for "the development of an effective voluntary health insurance or pre-payment program for the group over sixty-five with modest resources or low family income . . . (at physician compensation rates) which will permit the development of such insurance and pre-payment programs at a reduced premium rate."

Service Plan Rejected

Although the AMA request implied the desire for service plans rather than indemnity, Oklahoma Delegates previously (August 2) considered such a proposal and rejected it. It was the majority feeling at that time that such a concession, even on behalf of the elderly, would inadvertently lead the public to believe that the reduced fees would be an acceptable rate of pay for other special interest groups.

As a counter proposal, the Delegates elected to adhere to their traditional preference for indemnity-type programs and instructed the Committee on Insurance Programs for Senior Citizens to develop such a proposal, clear it through the Council, and re-submit it to them for final ratification. Under the indemnity concept, a low premium could be achieved and physicians could retain their right to apply their own means test with individual patients; thus, in the majority opinion, eliminate the abuses felt to be inherent in service contracts.

The Committee Reports

Immediately following the August meeting, the committee changed course as directed and began an investigation of an indemnity approach to the problem. Meetings were held on several occasions and the "Special 60" plan was devised in cooperation with Blue Shield representatives.

"Special 60" was approved by the Council on November 22 and referred to the House of Delegates.

Benefits Outlined

In a nutshell, the provisions of "Special 60" may be described by quoting the resolution passed jointly by the Blue Cross-Blue Shield Boards in response to the committee's request for action:

"RESOLVED, That the Blue Cross Board of Trustees approve the offering of the regular Blue Cross contract to people sixty years of age and over, except that the number of days' care be limited to thirty.

That the Blue Shield Board of Trustees approve the offering of the Blue Shield standard contract ($200 fee schedule) to people sixty years of age and over, except that in-hospital medical care be limited to eighteen days, beginning with the fourth day.

Further, that these contracts be offered in each county at the next regular non-group enrollment in that county; that the rate for these contracts shall be the same as that charged the regular non-group Blue Cross-Blue Shield members in that county.

Further, that the experience of holders of these contracts shall be allocated to the rest of the contract holders in that county, both group and non-group, on the basis of contracts in force."

In summary, the "Special 60" plan will correspond to the Standard 200 contract now in wide use throughout the state, except that the length of stay in the hospital has

been shortened from ninety days to thirty days, and the in-hospital medical care coverage has been reduced to eighteen days. The premium will be the same as the "200 contract" rate in a given county and will be subjected to the same county-rating system now being employed for all Blue Shield members. Sale of the "Special 60" will begin immediately, county by county, beginning on the regular non-group enrollment date for each county.

Council Meets Same Day

Although the Council had previously passed judgment on the insurance program, it never-the-less met on the morning preceding the House of Delegates meeting.

One of the highlights of the meeting was the Council's decision to launch a "crash program" to combat the Forand Bill, a measure now resting in Congress which would socialize medicine for some sixteen million social security recipients. After receiving first-hand information from Mr. Dick Nelson, AMA Field Service Division, the Council approved a report from the association's Federal Legislative Committee which called for the organization of twelve councilor district meetings on this subject during the first week of March.

Along the same line, the Council approved a motion calling for the re-statement of the association's opposition to the bill, and specifying that copies of this statement of policy be sent to members of Congress and the House Ways and Means Committee.

Another significant action of the Council was its decision to allocate $730 from the General Fund for the purpose of re-decorating the office of the Woman's Auxiliary, located in the OSMA Headquarters Building.

The average American family spent $294 for all personal health services (doctors, hospitals, drugs, etc.) in 1957-58, Health Information Foundation reports. But these costs were distributed unevenly. About 3 per cent of all families had no health costs; 31 per cent $1-$99; 34 per cent spent $100-$299; and the remaining 32 per cent spent $300 or more.

Kennedy, Liberals Push for Forand-Type Law

Representative Aime Forand (Democrat, R.I.), author of the infamous bill to socialize medicine for social security recipients, has found additional allies. Significantly among those piping the socialistic party line is presidential candidate Senator John F. Kennedy (Democrat, Massachusetts).

Serving as a member of the Senate Welfare Sub-Committee on Aging, labor-wooing Kennedy recently announced the inclusion of social security health benefits in his political platform. According to Kennedy and subcommittee cohorts, "The number one problem of America's senior citizens is how to meet the costs of health care at a time when income is lowest and potential or actual disability is highest."

Beside Kennedy, Chairman Pat McNamara (Democrat, Michigan), and Senator Joseph S. Clark (Democrat, Pennsylvania) and Jennings Randolph (Democrat, W. Virginia) joined in the report which also called for a big increase in monthly social security benefits. The disturbing development came after an alleged survey of the needs of the aging which the barnstorming group conducted in eight cities across the nation. Opponents of the Forand Bill, the Kennedy proposal and other similar legislation have correctly observed that Kennedy, color-bearer for the group, was seldom present at any of the public hearings which were staged. Had he been there, it is also observed that his legally-trained mind would have noticed that the conduct of the meetings, coupled with the prejudices of Chairman McNamara, branded the information obtained as biased, political ammunition, rather than a factual, objective report of the true conditions.

The report said that about eight million Americans over age sixty-five, half of the present total, "cannot afford today, decent housing, proper nutrition, adequate medical care, preventive or acute, or necessary recreation." Among those taking exception to these loosely-drawn figures, are the minority members of McNamara's sub-commit-

tee, Senators Everett M. Dirksen (Republican, Illinois) and Barry Goldwater (Republican, Arizona). They have offered a minority report which supports voluntary enterprise as the best method of meeting the problems of the aged, and they are more conservative in their estimate of need.

Oklahoma Figures Differ

On the Oklahoma scene, the estimates of the needy-aged run closer to 10% of the total population over sixty-five. Nearly one-half of this age group in the state is covered by the Medical Care Program for Public Assistance Recipients. More than 10% have Blue Cross plans, and another 10% or more have commercial health insurance. When the financially independent, VA beneficiaries and others are subtracted from the remainder, the balance of "unknowns" falls considerably short of the "one-half" alleged to be medically indigent.

Forand Bill Fulminates

Meanwhile, back at the House Ways and Means Committee, things were popping too. Special field men of the AF of L-CIO, hired expressly to drum up pressure for the passage of the Forand Bill, were applying the leverage to conservative-minded Chairman Wilbur Mills to get the bill reported favorably out of committee and to the floor of the House.

In addition, Secretary of Health, Education and Welfare Arthur Fleming was wavering from his previous opposition to government intervention in the health care field. The administration is now reported to be drafting a counter-proposal to the Forand Bill which will also be introduced through the Ways and Means Committee along with other amendments to the Social Security Act.

Taxpayers Protest

In Oklahoma, as in all other states, medical leadership has inspired physicians and other thinking-taxpayers into vociferous opposition to Forand-Type legislation. Great efforts have been made to match labor's write-in volume and neutralize the pressure being applied on Ways and Means Committee members and other U.S. Representatives. State and County medical societies across the land are sending official resolutions to their Congressmen and to Wilbur Mills. Other organizations have allied themselves with medicine and are making similar, concerted efforts to achieve representative government.

Special bulletins have been mailed to every physician in the state, asking him to write his congressman (and Mills) and to get ten of his friends and patients to do the same. The OSMA Federal Legislative Committee met on February 25 with leaders of other statewide organizations and received their pledges of cooperation in launching write-in campaigns.

To further incite the voice of freedom, the Federal Legislative Committee organized OSMA Councilor District meetings and sent speakers and materials all over the state.

According to J. R. Stacy, M.D., Chairman of the committee, "the threat of this type legislation has never been more imminent. Both Republicans and Democrats have recognized the power of the aged voting bloc, and, right or wrong, the course of least resistance may be followed if the taxpayers don't speak loud, long and continuously for a sound dollar and a solvent government!

Voluntary enterprise is solving what problems there are that now confront a certain segment of our senior citizens . . . but simply resting on laurels will not get the job done. Congressmen must be told over and over again of their supreme obligation to stop give-away programs and restore our economy to a sensible balance. Promiscious foreign and domestic spending and the devaluation of the dollar which accompanies it are the real villains of the elderly . . . and of anyone else trying to live on a fixed income in an inflationary world. The unnecessary expenditure of $2 billion for the Forand Bill would only stoke the flame . . . and further expansion of the program to keep up with inflation would be absolutely predictable."

Physicians Play Leading Role in Aged Study

Over sixty members of the Oklahoma State Medical Association will take active parts in the Oklahoma phase of the White House Conference on Aging. The physicians are now serving in various committee capacities in a statewide survey of the status of the aging and aged populations of Oklahoma, preparatory to a state conference in June, 1960, and a national conference in January, 1961.

Inspired by OSMA

Oklahoma's participation in the national project and the University of Oklahoma's guidance of the state phase of the program are the results of action taken by the association's Committee on Aging nearly a year ago. At that time, it was learned that $15,000 federal grants were being made available to states under the provisions of Public Law 85-908, the "White House Conference on Aging Act."

The Act further provided that states should use these funds to survey the status of the aging on a state-by-state basis, conduct state conferences and be prepared to formulate and submit to the national conference certain recommendations . . . "for action in improving and developing programs to permit the country to take advantage of the experience and skills of older persons, to create conditions which will better enable them to meet their needs, and to further research on aging."

The association committee, chaired at that time by Hayden Donahue, M.D., anticipated that the Oklahoma survey funds would be sought by special interest groups, and recommended to the Governor that application for such funds should be made on behalf of the Extension Division of the University of Oklahoma, where an unbiased study could most likely be made. The Governor accepted this recommendation and appointed O.U. President George L. Cross as Chairman of the Oklahoma Committee on Aging.

Four Take Roles of Leadership

OSMA President Alfred T. Baker, M.D., John W. DeVore, M.D., Walker Morledge, M.D., and Lee Honska, M.D., have accepted appointments of primary responsibility in the state organization. Doctor Baker is serving as a member of Doctor Cross' Executive Committee and Doctor DeVore is Chairman of the fourteen fact-finding committees which will gather the bulk of the information upon which the Oklahoma recommendations will be made. Doctor Morledge is Chairman of the all-important Fact-Finding Committee on Health and Doctor Honska is Chairman of the Committee on Research, Training and Demonstration.

Health Aspects

Physicians serving with Doctor Morledge on the health committee are: John F. Burton, M.D., Donald W. Branham, M.D., Robert C. Lowe, M.D., and Forest R. Brown, M.D. To adequately cover the subject, the committee is divided into three sub-committees. Doctor Burton chairs the Sub-Committee on Financing Health Care; Doctor Branham heads the Sub-Committee on Health Needs of the Aging; and Doctors Brown and Lowe are co-chairmen of the Sub-Committee on Health Care Resources.

Other Committees

In addition to the physician-dominated Fact-Finding Committee on Health, Oklahoma medics are well represented on the remaining thirteen fact-finding groups. Serving as committee members are: Frank L. Adelman, M.D., Enid; John A. Bealor, M.D., Norman; Don Bernamonti, M.D., Muskogee; Donald W. Branham, M.D., Oklahoma City; Rheba H. Edwards, M.D., Oklahoma City; Shelby G. Gamble, M.D., Okmulgee; K. H. Geocaris, M.D., Tulsa; A. A. Hellams, M.D., Oklahoma City; Herbert B. Hudnut, Jr., M.D., Oklahoma City; Earl D. McBride, M.D., Oklahoma City; E. C. Mohler, M.D., Ponca City; K. W. Navin, M.D., Shawnee; T. A. Ragan, M.D., Norman; Louis H. Ritzhaupt, M.D., Guthrie; S. C. Shepard, M.D., Tulsa; and J. R. Stacy, M.D., Oklahoma City.

Mayors' Committees

Another important fact-finding feature of the state survey is the employment of mayors' committees. Again, the medical profession is well represented. On the first thirty-five mayors' committees formed, thirty-seven physician names appeared, five of whom were appointed as chairmen.

'Something For Everyone' At Annual Meeting

A diversified scientific and social program and an outstanding array of guest speakers will be offered Oklahoma physicians at the 54th Annual Meeting of the Oklahoma State Medical Association, to be held in Oklahoma City, May 2, 3, and 4, 1960. Newer concepts in medical and surgical techniques will prevail throughout the fifteen individual scientific lectures and the three scientific symposia which are scheduled for the meeting.

The complete program and details of the 54th Annual Meeting will appear in the April issue of the Journal.

Despite emphasis on "newer concepts," all scientific events have been planned with an eye on practicality. According to Doctors C. M. Bielstein and Bert F. Keltz, General Chairman and Scientific Works Chairman respectively, the program has been keyed to appeal to specialists and general practitioners alike.

Rounding out the contributions to be made by the fifteen guest speakers, Oklahoma physicians will also be treated to a high-quality display of scientific and educational exhibits, plus a full calendar of related activities and social events.

Speakers Named

Listed as guest speakers are: Edgar V. Allen, M.D., Internal Medicine, Rochester, Minnesota; Nadene Coyne, M.D., Physical Medicine, New York, New York, Grayson Carroll, M.D., Urology, St. Louis, Missouri; George Deaver, M.D., Physical Medicine, New York, New York; Michael DeBakey, M.D., Surgery, Houston, Texas; Vincent T. Derbes, M.D., Dermatology, New Orleans, Louisiana; Eugene L. Derlacki, M.D., E.E.N.T., Chicago, Illinois; Murdina Desmond, M.D., Pediatrics, Houston, Texas; Benjamin Felson, M.D., Radiology, Cincinnati, Ohio; Schuyler Kohl, M.D., Obstetrics and Gynecology, Brooklyn, New York; Mr. Ken Rawlinson, Collegiate Athletic Trainer, Norman, Oklahoma; Harold Rosen, M.D., Psychiatry, Baltimore, Maryland; Mr. Gerald J. Skibbins, Public Opinion Research, Princeton, New Jersey; Harold A. Sofield, M.D., Orthopedic Surgery, Oak Park, Illinois; and Mario Stefanini, M.D., Internal Medicine, Brighton, Massachusetts.

Members of the OSMA who will participate are: Shelby Gamble, M.D., Ella Mary George, M.D., William Jaques, M.D., Herbert Kent, M.D., Earl McBride, M.D., Tom Points, M.D., Carl Lindstrom, M.D., G. R. Russell, M.D., and J. R. Stacy, M.D.

Public Opinion Expert Featured

A change of pace from the scientific format will be the equally important talk to be given by Mr. Skibbins, Research Associate of the Opinion Research Corporation of Princeton, New Jersey. Scheduled as the luncheon speaker for Tuesday, May 3, he will discuss the timely topic, "How Public Opinion Can Socialize A Profession or Industry."

In an advance outline of his talk, the public opinion expert reported that recent attitude research has revealed the effects of public opinion on broad social legislation such as labor law, government electric power and socialized medicine. A further definition of this process indicates that the medical profession is now an incidental target of such a movement, to the extent that effective counter action must be taken soon if the adverse trend of the opinion movement is to be stopped in time.

Panels Planned

On Tuesday morning, May 3, Doctors Allen, Stefanini and DeBakey will present papers concerning their particular interests in the subject, "Newer Concepts and Developments in Peripheral Vascular Disease." Doctor Allen will lead off with a discussion pertaining to the natural history of arteriosclerosis obliterans. He will be followed by Doctor Stefanini who will speak on recent findings regarding blood clotting. Lastly, Doctor DeBakey will talk about recent de-

velopments in the surgical approach to the disease.

Following the formal presentations, the three physicians will participate in a panel discussion, moderated by William Jaques, M.D., Oklahoma City pathologist.

During Tuesday afternoon, the second featured panel will be presented. Doctors Kohl and Desmond will first deliver papers on the subject of "Perinatal Mortality," speaking from the standpoints of the obstetrician and pediatrician. These lectures will be followed by presentations from Doctors Points, Lindstrom and Russell, who will discuss work being done locally in this field of interest. Time will then be allotted for a question and answer session, moderated by Doctor Kohl.

Wednesday morning, the closing session of the scientific program, will be devoted exclusively to the important subject of rehabilitation, with particular emphasis on hemiplegia and paraplegia. This presentation, which will include panel discussions and clinical demonstrations, is now being planned by Doctors Deaver and Coyne, from N.Y.U.'s Bellevue Medical Center, in cooperation with Shelby G. Gamble, M.D., Okmulgee.

Prizes for Scientific Exhibits

Nearly forty physicians and organizations are expected to compete for awards in the Scientific Exhibits Section. An increasingly popular part of the annual meeting, the exhibit section will have added interest this year with the development of the "Klinical Kaleidoscope," a section devoted to a collection of bizarre or unusual cases. The inauguration of this feature is expected to appeal to many physicians who would like to illustrate one of their interesting cases rather than preparing a booth on original research or a series of cases. The bizarre cases will be reviewed by the Editorial Board of *The Journal* for possible publication.

Applications Due April 1

Booths will not be de-emphasized, however, and an excellent response is expected in this area. Applications will be received by the Scientific Exhibits Committee up to April 1. The Form to be used is printed on page 190 of this *Journal*.

Other Highlights

Scientific activities will be proportionately blended with social events and other activities in an effort to offer participants a well-balanced meeting.

The President's Inaugural Dinner-Dance is scheduled for Tuesday evening, May 3, in the Persian Room of the Skirvin Tower Hotel. President Alfred T. Baker, M.D., Durant, will culminate a busy year as the association's top executive officer when he turns over the gavel to Walter E. Brown, M.D., Tulsa.

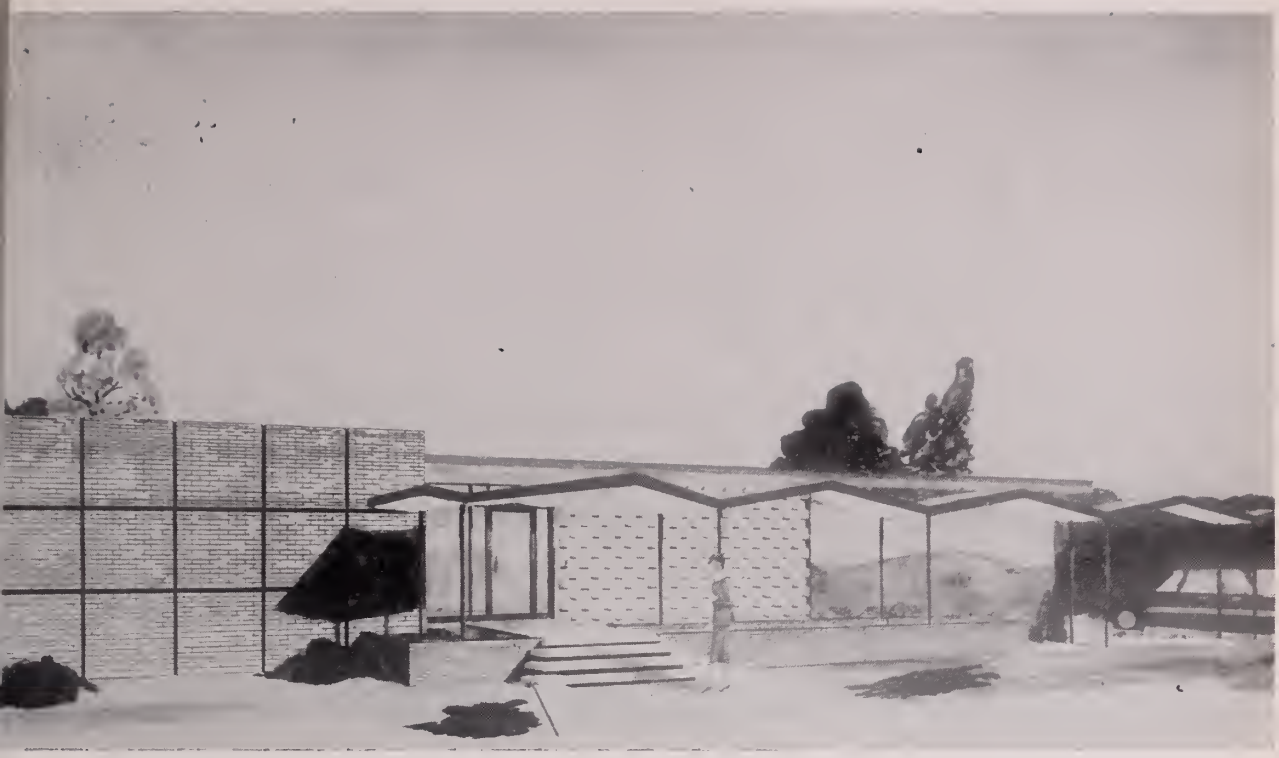
The Annual Physicians Hobby Show will be held continuously during the meeting, under the sponsorship of the Woman's Auxiliary. The varied pastime talents of Oklahoma medics will be on display in the Regency Room of the Skirvin Hotel. Those wishing to contribute samples of their leisure activities may apply for exhibit space by completing the application on page 196 of this issue of *The Journal*.

Order Anti-Forand Pamphlets

A new AMA pamphlet, "Political Medicine Is Bad Medicine," may be ordered in quantity by physicians for distribution to their patients.

The pamphlet contains eleven questions and answers about the controversial Forand Bill. It's size makes it suitable for a letter or statement stuffer, or it would be excellent reading material for the waiting room.

Since the Forand Bill poses a serious threat to the private practice of medicine, as well as to the physical and economic health of the nation, the brochures are being offered free to physicians. Order yours now by writing: The American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois.



Muskogee To Have New Office Building

Now being constructed at 201 South 36th Street in Muskogee, is the new office building of Perry Hewitt, M.D. Site of the building borders a park, as does the location of the new Muskogee General Hospital.

Exterior of the 3,420 square foot building is of brick and enamel trim, with green tinted glass block and white case stone combined in a pattern for the exterior wall of the lobby. Interior walls are lightweight concrete block, many of which are constructed to form a decorative pattern. The modern structure will feature a covered entrance and ramped walkway for aged, ambulatory patients.

Built-in desks and cabinets are plastic laminate covered to give life-time service. The interior decoration will be a combination of patterned walls, acoustical plaster ceiling, vinyl floors and modern furnishings.

A brass and walnut partition of cabinets separates a conference room and examination room which has been planned for Doctor Hewitt's suite. A reception room is separated from the office waiting area by a paneled divider of glass, wood-grained plastic laminate and brass supports.

Rental suites will be developed to the requirements of the individual tenants with many of the decorative features being repeated throughout. The suites can be made into special purpose rooms with partitions that can be removed and rearranged, yet give the appearance of permanence and be soundproofed. All tenants will have private entrances into their suites and have access to the library. Individual air conditioning units and adequate electrical service for such items as X-ray machines, etc. will be available.

Off street parking has been provided to the rear and one side of the building. Designed to require the simplest maintenance, the structure will be adaptable to changes in layout for varying medical practices, and include future additions to the building when desired.

Architects for the office building are Zaroor and Davis, Muskogee.

Program Completed for Third Oklahoma Colloquy

Devoted to Adrenal Steroids, the Third Oklahoma Colloquy on Advances in Medicine, developed by the Department of Medicine and Division of Postgraduate Education, University of Oklahoma School of Medicine, will be held in the school auditorium March 24, 25, 26, 1960. Collaborating societies are the Oklahoma City Internists Association, the Oklahoma City Surgical Society and the Oklahoma City Pediatric Society. Sponsor for the three day meeting will be Merck Sharp and Dohme.

Registration fee for the colloquy is $35.00 for the entire program or $15.00 per day. A complete program follows:

THURSDAY, MARCH 24

8:00 a.m. Registration

Morning Session

8:30 a.m. Announcements

8:45 a.m. INTRODUCTION
Stewart G. Wolf, Jr., M.D.
Moderator: Allen R. Hennes, M.D.

8:50 a.m. SOME IDEAS BEHIND THE SYNTHESIS OF THE ANTI-INFLAMMATORY STEROIDS
Lewis H. Sarett, Ph.D.

9:30 a.m. THE ROLE OF THE ADRENAL CORTEX IN THE ETIOLOGY OF DISEASE
Dwight J. Ingle, Ph.D.

10:20 a.m. Intermission

10:30 a.m. RECENT ADVANCES CONCERNING THE ADRENOCORTICAL SECRETION
Oscar Hechter, Ph.D.

11:10 a.m. THE PRINCIPLES OF ADRENOCORTICOSTEROID THERAPY: SYSTEMIC, PARENTERAL, INTRA-ARTICULAR AND LOCAL ADMINISTRATION
Richard T. Smith, M.D.

11:50 a.m. Discussion.

Afternoon Session

Moderator: W. O. Smith, M.D.

1:30 p.m. THE MECHANISMS CONTROLLING ADRENAL CORTICAL SECRETION IN RESPONSE TO TRAUMA
David M. Hume, M.D.

2:10 p.m. THE ROLE OF THE ADRENALS AND THE ADRENAL STEROIDS IN ELECTROLYTE AND FLUID METABOLISM
Louis J. Soffer, M.D.

2:50 p.m. PATHOPHYSIOLOGY OF ALDOSTERONE SECRETION IN MAN
S. Richardson Hill, Jr., M.D.

3:30 p.m. Intermission

3:45 p.m. PHARMACOLOGIC INHIBITION OF ADRENAL SECRETION AND ACTION
Don H. Nelson, M.D.

4:25 p.m. Discussion

FRIDAY, MARCH 25

Morning Session

Moderator: P. C. Johnson, M.D.

8:30 a.m. DIAGNOSIS AND TREATMENT OF THE ADRENOGENITAL SYNDROME
Lawson Wilkins, M.D.

9:10 a.m. A STUDY OF CUSHING'S SYNDROME
Louis J. Soffer, M.D.

9:50 a.m. THE ADRENAL CORTEX AND LIPID METABOLISM
R. Palmer Howard, M.D., Robert H. Furman, M.D.

10:15 a.m. Intermission

10:25 a.m. PLASMA ACTH
Don H. Nelson, M.D.

11:05 a.m. THE PREVENTION AND TREATMENT OF UNDESIRABLE REACTIONS FROM ADRENAL STEROID THERAPY
Richard T. Smith, M.D.

11:45 a.m. Discussion

Afternoon Session

Moderator: Martin Cummings, M.D.

1:30 p.m. THE USE AND HAZARDS OF ADRENAL STEROIDS IN TUBERCULOSIS
Harold Muchmore, M.D.

1:50 p.m. THE USE OF STEROIDS IN INFECTIONS
Ralph Tompsett, M.D.

2:30 p.m. THE PRODUCTION AND DEGRADATION OF CORTISOL IN HUMAN BACTEREMIC SHOCK
James C. Melby, M.D.

3:00 p.m. Discussion

3:30 p.m. Intermission

BASIC SCIENCES SECTION

Room 120 Medical School

Moderator: Mark R. Everett, Ph.D.

3:45 p.m. THE EFFECT OF SOME STRESSORS ON SYMPTOMS OF CORTISONE OVERDOSAGE
Dwight J. Ingle, Ph.D.

4:30 p.m. CONCERNING THE MODE OF DESOXY CORTICOSTERONE ACTION AT THE CELLULAR LEVEL
Oscar Hechter, Ph.D.

5:15 p.m. Discussion

MEDICINE SECTION

Oklahoma City Internist Association
Medical School Auditorium

Moderator: Robert Bird, M.D.

3:45 p.m. PATHOLOGIC AND CLINICAL OBSERVATIONS IN STEROID TREATED ULCERATIVE COLITIS
Howard M. Spiro, M.D.

4:10 p.m. ADRENAL CORTICAL SECRETORY ACTIVITY IN RHEUMATIC DISEASE STATES
S. Richardson Hill, Jr., M.D.

4:35 p.m. THE USE OF ADRENAL STEROIDS IN LEUKEMIAS AND LYMPHOMAS
William J. Harrington, M.D.

5:05 p.m. THE ROLE OF ADRENAL STEROIDS AND OF ENDOCRINE ABLATION IN THE MANAGEMENT OF NEOPLASIA
L. P. Eliel, M.D.

5:30 p.m. Discussion

PEDIATRICS SECTION

Oklahoma City Pediatrics Society
Room 104-G, Childrens Hospital
Moderator: Harris D. Riley, Jr., M.D.

4:00 p.m. ABNORMALITIES OF SEX DIFFERENTIATION: ETIOLOGY, DIAGNOSIS, SELECTION OF SEX AND TREATMENT
Lawson Wilkins, M.D.

4:40 p.m. THE USE OF ANTI-INFLAMMATORY STEROIDS IN PEDIATRICS
C. William Daeschner, Jr., M.D.

SURGERY SECTION

6:30 p.m. Evening meeting with Oklahoma City Surgical Society, Glen's Hick'ry Inn, 2815 N.W. 10th Street

8:30 p.m. THE USE OF ADRENAL STEROIDS IN THE SURGICAL TREATMENT
David M. Hume, M.D.

6:30 p.m. DINNERS
1. Basic Science Dinner
Thunderbird Motel
2. Oklahoma City Internist Association
Faculty House—601 N.E. 14th Street
3. Oklahoma City Surgical Society
(See Surgery Section above)
4. Oklahoma City Pediatric Society
Skirvin Hotel

SATURDAY, MARCH 26

Morning Session

Moderator: Leon Freed, M.D.

8:30 a.m. THE USE OF ANTI-INFLAMMATORY STEROIDS IN THE CONTROL OF COLLAGEN DISEASES
Bram Rose, M.D.

9:10 a.m. USES OF ADRENAL STEROIDS IN RENAL DISEASE
C. William Daeschner, Jr., M.D.

9:50 a.m. THE USE OF ADRENAL STEROIDS IN IMMUNOHEMATOLOGIC DISORDERS
William J. Harrington, M.D.

10:30 a.m. Intermission

10:45 a.m. PHYSIOLOGIC AND CLINICAL IMPLICATIONS OF THE STEROID-INDUCED PEPTIC ULCER
Howard M. Spiro, M.D.

11:25 a.m. Discussion

Afternoon Session

Moderator: Carl Smith, M.D.

1:35 p.m. THE USE OF THE ANTI-INFLAMMATORY STEROIDS IN THE MANAGEMENT OF ALLERGY
Bram Rose, M.D.

2:10 p.m. AN EVALUATION OF PITUITARY SUPPRESSION IN STEROID THERAPY WITH THE USE OF BACTERIAL PYROGEN
James C. Melby, M.D.

2:40 p.m. Intermission

2:55 p.m. THE RELATIVE POTENCIES OF STEROIDS
Kelly M. West, M.D.

3:25 p.m. THE RECOGNITION AND MANAGEMENT OF ADRENAL INSUFFICIENCY
Allen R. Hennes, M.D.

3:55 p.m. Discussion

Appearing on the program as guest speakers will be:

C. Wm. Daeschner, Jr., M.D., Associate Professor of Pediatrics, Baylor University College of Medicine, Houston, Texas; William J. Harrington, M.D., Associate Professor of Medicine, Washington University School of Medicine, St. Louis, Missouri; Oscar Hechter, Ph.D., Research Associate Professor, School of Medicine (Boston), Worcester Foundation for Experimental Biology, Shrewsbury, Massachusetts; S. Richardson Hill, Jr., M.D., Associate Professor of Medicine, Medical College of Alabama, Birmingham, Alabama; David M. Hume, M.D., Chairman, Department of Surgery, Medical College of Virginia, Richmond, Virginia; Dwight J. Ingle, Ph.D., Chairman, Dept. of Physiology, University of Chicago, Chicago, Illinois; James C. Melby, M.D., Assistant Prof. of Medicine and Assistant Prof. of Biochemistry, University of Arkansas School of Medicine, Little Rock, Arkansas; Don H. Nelson, M.D., Associate Professor of Medicine, University of So. California, Los Angeles, California; Bram Rose, M.D., Associate Professor of Medicine, McGill University Faculty of Medicine, Royal Victoria Hospital, Montreal, Canada; Lewis H. Sarett, Ph.D., Director, Synthetic Organic Chemistry, Merck Sharp & Dohme Research Laboratories, Rahway, New Jersey; Richard T. Smith, M.D., Associate Professor of Medicine, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania; Director of the Department of Rheumatology, Benjamin Franklin Clinic, Philadelphia, Pennsylvania; Director, Medical Services, Merck Sharp & Dohme Research Laboratories, West Point, Pennsylvania; Louis J. Soffer, M.D., Clinical Professor of Medicine, State University of New York, Attending Physician and Head of Endocrinology, The Mount Sinai Hospital, New York, New York; Howard M. Spiro, M.D., Assistant Professor of Medicine, Yale University School of Medicine, New Haven, Connecticut; Ralph Tompsett, M.D., Director of Medical Education, Baylor University Medi-

cal Center, Dallas, Texas; and Lawson Wilkins, M.D., Professor of Pediatrics, The Johns Hopkins Hospital, Baltimore, Maryland.

Participants from the University of Oklahoma Medical Center will be:

Robert M. Bird, M.D., Associate Professor, Department of Medicine and of Physiology; Martin Cummings, M.D., Professor and Chairman, Department of Microbiology; L. P. Eliel, M.D., Research Professor of Medicine, Director, Oklahoma Medical Research Foundation; Mark R. Everett, Ph.D., Director and Dean, University of Oklahoma Medical Center; Leon C. Freed, M.D., Assistant Professor of Medicine; Robert H. Furman, M.D., Research Associate Professor of Medicine, Cardiovascular Section, Oklahoma Medical Research Foundation; Allen R. Hennes, M.D., Assistant Professor of Medicine, *Assistant Chief Medical Service; R. Palmer Howard, M.D., Research Associate Professor of Medicine, Cardiovascular Section, Oklahoma Medical Research Foundation; Phillip C. Johnson, M.D., Associate Professor of Medicine, *Chief, Radioisotope Service; Harold Muchmore, M.D., Assistant Professor of Medicine, *Chief of Tuberculosis and Infectious Disease Section; Harris D. Riley, Jr., M.D., Professor and Head, Department of Pediatrics; Carl Smith, M.D., Instructor in Medicine, Assistant Chief of Medicine, Central State Hospital; W. O. Smith, M.D., Assistant Professor of Medicine, *Assistant Chief, Radioisotope Service; Kelly M. West, M.D., Assistant Professor of Medicine; and Stewart G. Wolf, M.D., Professor and Head Department of Medicine.

*Veterans Administration Hospital

Medicare Identification System Outlined

The Department of Defense has acknowledged the difficulty physicians have in obtaining Identification Cards from some Medicare patients. In a recent letter to Blue Shield, the D.O.D. outlined certain alternate procedures possible in cases where the Identification Card (D.D. Form 1173) is not available at the time of care.

In emergency cases only, the Medicare claim form must still be accompanied by a statement from the physician certifying that the case was an emergency. In addition, the D.O.D. suggests the following types of collateral identification of the patient:

1. A statement by a local commander establishing the patient's eligibility during the period of care.
2. Statement by any official of a uniformed service.
3. A statement by the physician or hospital authority that he has personal knowledge of the patient's eligibility.
4. A Social Security or driver's license number that identifies the patient should be entered in the space designated on the claim form for the Identification Card. However, when this type of identification is used, it is important that the identification document reveal the same name as that which is signed to the claim form.

As in the past, when an Identification Card number is not submitted with a claim, the reason why this card was not available must be stated by the claiming physician.

In non-emergency cases, when the Identification Card is not readily available, the patient is considered only a *potential* beneficiary of the Medicare program until eligibility has been definitely established. Payment of the physician's claim must be contingent upon the claim being documented by an authorized military official.

The letter also indicated a complete change in policy toward cards that bear, (a) an expired date of eligibility , (b) no date, or (c) an "indefinite" date. Blue Shield (Fiscal Agent) may *not* process a physician's claim on which the eligibility of the patient is marked as such. The claim *must* be supported by some authorized military official statement that the patient was eligible during the period of care covered by the claim.

In general, this latest letter from the Department of Defense re-emphasizes the importance and need for proper *identification* of a Medicare patient receiving civilian care. Physicians are urged to use all possible care in obtaining and relaying correct information regarding the patient's identity. It should be noted that this letter does *not* in any way change present *policies* regarding the "permit" system which establishes the patient's *eligibility* for civilian care where military facilities are not available. However, so-called "permit" is now referred to as a "Non-Availability Statement" (form D.D. 1251).

Southwest Allergy Forum Will Meet in Oklahoma City

Oklahoma City's Skirvin Hotel will be the site of the 21st Annual Allergy Forum to be held April 2, 3, 4, and 5, 1960. Forty-five guest speakers and panelists will highlight the diversified program which has been planned.

Founded twenty-one years ago in San Antonio, Texas by a small group of allergists who met to discuss their common problems in a round table-type meeting, the organization has now grown to a registration of over two hundred and fifty at annual meetings. Participants attend from all parts of the United States and Mexico, taking an active part in the discussions.

The specialty of allergies is only thirty years old, but there are approximately one thousand specialists in this field which now has two national organizations.

Subjects for panel discussions will include: Pulmonary Ventilation, Steroids, Stump the Experts—Questions and Answers, and Rare, Unusual and Personal Problems in Allergy. Round table discussions will embrace Allergic Skin Disorders, Allergy of the Nose, Bronchial Asthma, Urticuria, Allergic Headaches, Food Allergy and Gastro-Intestinal Allergy. Individual presentations will be given on "Cryptococcus as an Aero-Allergen," "Occupational Bronchial Asthma Due to Inhalent Aspergillus Sensitivity," "Mold Problems in Geographic Area 11—The Southwest," "Lichen Causes Dermatitis" and "Molds and Bacteria in the Etiology of Respiratory Allergic Diseases XXI."

Out-of-State Speakers

Listed either as speakers, panelists or participants in round table discussions are the following out-of-state guests: Marie B. Morrow, Ph.D., Austin, Texas; George H. Meyer, M.D., Austin, Texas; Aaron Weiner, M.D., Fair Lawn, New Jersey; Boen Swinny, M.D., San Antonio, Texas; Rita Don, M.D., El Paso Texas; James Holman, M.D., Dallas, Texas; Warren J. Raymer, M.D., Houston, Texas; Norris Beasley, M.D., Fort Lauderdale, Florida; Homer E. Prince, M.D., Crockett, Texas; John Affeldt, M.D., Los Angeles, California; Clifford H. Kalb, M.D., Milwaukee, Wisconsin; Samuel C. Bukantz, M.D., Denver, Colorado; Stanislaus H. Jaros, M.D., Harlingen, Texas; Alexander Roth, M.D., Prairie Village, Kansas; Maurice Barnes, M.D., Waco, Texas; W. C. Service, M.D., Colorado Springs, Colorado; A. C. Stutzman, M.D., Saint Louis, Missouri; Allan Cazort, M.D., Little Rock, Arkansas and Kenneth L. Craft, M.D., Indianapolis, Indiana.

Also participating will be: William C. Grater, M.D., Dallas, Texas; R. Dale Dickson; M.D., Memphis, Tennessee; Jonathan Foreman, M.D., Columbus, Ohio; Clyde F. Elkins, M.D., Lubbock, Texas; Walker L. Rucks, M.D., Memphis, Tennessee; H. Whitner Boggs, M.D., Shreveport, Louisiana; Carl Marsh, M.D., Memphis, Tennessee; Theophilus Painter, M.D., Austin, Texas; Sim Hulsey, M.D., Fort Worth, Texas; George Gwen, M.D., Jackson, Mississippi; Cecil Kohn, M.D., Kansas City, Missouri; Tom Johnston, M.D., Little Rock, Arkansas; A. Ford Wolfe, M.D., Temple, Texas; Larry Halpin, M.D., Cedar Rapids, Iowa; Roy E. Schirmer, M.D., Fort Smith, Arkansas; Frank Joyce, M.D., Denver, Colorado; Henry Ogden, M.D., New Orleans, Louisiana; Harry Lasar, M.D., Wichita, Kansas; Thomas R. McElhenney, M.D., Austin, Texas; Robert Willard, M.D., Boulder, Colorado; Ralph Hale, M.D., Wichita, Kansas; Truman C. Terrell, M.D., Fort Worth, Texas; Frank English, M.D., Roswell, New Mexico; Charles W. Vivian, M.D., Phoenix, Arizona; and George Seibold, M.D., Wichita Falls, Texas.

Two laymen who will appear as panelists are W. A. Gardner, Engineer, Duncan Oxygen Therapy Company, Duncan, Oklahoma and Fred Schnitz, Puritan Compressed Gas Company, Kansas City, Missouri.

Local Participants

Oklahoma City physicians who will appear on the program will be Dick H. Huff, M.D., Henry H. Turner, M.D., Richard W. Payne, M.D., Robert Morgan, M.D., L. Chester McHenry, M.D., and Stewart Wolfe, M.D.

Arrangements for the Forum have been made by the Executive Committee comprised

of Johnny A. Blue, M.D., President, George L. Winn, M.D., Secretary, Fannie Lou Leney, M.D., Treasurer, Dick H. Huff, M.D., Chairman of the Program Committee and Robert E. Ellis, M.D., Chairman of Exhibits. Oklahome City Allergists who have attended the Executive Committee in planning the Forum are Carrol Pounders, M.D., Phillip M. McNeill, M.D., Vernon D. Cushing, M.D. and Charles E. Delhotal, M.D.

Eighteen exhibitors who are sponsoring the forum will have exhibits during the meeting.

Special entertainment for the ladies will include a "Bird's Eye View of the City," a tea, a visit to the Oklahoma City Art Center and luncheon at the Surrey House in Hummel Village.

Drug Makers Refute Charges

Doctor Austin Smith, former Editor of the *AMA Journal* and now President of the Pharmaceutical Manufacturers Association, told Senate investigators on February 23 that Americans "would be paying a billion dollars a year more for drugs if the price of medicine in the past few years had gone up only as much as the total cost of living."

He said the drug industry has "an outstanding record of low prices maintained in an inflationary period." Doctor Smith testified before the Kefauver Anti-trust Committee in rebuttal to the Senator's criticisms of industry practices.

He declared, furthermore, that "on the basis of the record," he knows of "no other American industry that has contributed more from its resources to the public welfare."

Modern drugs have helped to add nearly ten years to the lifespan of the average American within the past thirty years, he observed, adding: "since 1947 this industry has spent about $1 billion in research alone."

He said the pharmaceutical industry has served the public interest by driving prices down to the lowest possible levels.

Scoring earlier criticism of industry prices, he said there is "grave danger of misinforming the public when attention is directed toward the single product of a single company . . . without relating these to the many thousands of products produced by the entire industry."

Doctor Smith declared that the competitive pharmaceutical industry in America "has played a role unparalleled elsewhere in the world in reducing human suffering and in helping to eradicate diseases which have crippled mankind for centuries."

Citing the lack of inventiveness in drugs within the Soviet Union, he emphasized that "there has been no missile gap in pharmaceutical research."

Recalling major breakthroughs attributable in significant ways to U.S. drug industry laboratories, Doctor Smith named the discovery and development of the sulfa drugs, the synthesis and discovery and development of high-potency corticosteroids, the mass production of penicillin, and the discovery and development of the other broad-spectrum antibiotics.

Emphasizing the "staggering investment" which the drug industry puts into research, Doctor Smith said that "the chance that any given research involving a new potential medicine will be successful stands at 2,865 to one against the manufacturer."

He said the industry's research resulted in valuable palliative treatment for cancer, and that the industry was also responsible for the discovery and development of tranquilizer drugs for treating mental illness, oral diabetic drugs, and the hydrazide derivatives for treating tuberculosis.

Doctor Smith said that every major advance of the U. S. drug industry has been accompanied by "astronomical dollars-and-cents savings to the American people."

He said that vast reductions in medical expenses were made possible by rapid cures for illnesses and by the shorter length of hospitalization which results in increased productivity of those to whom new drugs have made cure possible.

Doctor Smith said that this has also meant "mammoth tax savings to the public" by reducing the cost of public health services.

Federal Health Act; A Significant Step

The Federal government, as the largest employer in the U.S.A.—is about to provide medical care security to some 4 million Federal employees and their dependents.

Under the new Health Benefits Act passed by Congress last September, government workers will begin to enroll about June 1, 1960 in one or another of four types of hospital and medical care programs: 1) a service benefit plan (Blue Cross-Blue Shield); 2) an indemnity plan (underwritten by an insurance company); 3) an employee organization plan (of which a considerable number have been set up by Federal employee organizations); 4) a comprehensive "closed panel" plan (such as the Kaiser Health Plan or H.I.P.)—where such programs exist. Federal contributions will commence in July toward the cost of whatever plan may be selected by each Federal worker.

Each employee will have the utmost freedom to choose among the specific plans to be approved by the U. S. Civil Service Commission in negotiations now going on between the Commission and the "carriers" of the four types of programs specified in the Act.

The government has shaped its program in accordance with the mutual desire of its employees and their doctors for a free choice of physician and plan.

To meet the natural requirements of the Civil Service Commission for a reasonable degree of uniformity among the programs offered by the 79 Blue Cross and the 67 Blue Shield Plans, many Plans will have to alter or add to their established benefit provisions. This will call for cooperation among all who are providing services to patients under our local Blue Shield Plans.

The significance of the Federal Employee Health Benefits Act for the future of American medicine can scarcely be exaggerated. Under the terms of this act, government will contribute toward the cost of a hospital and medical care coverage program for all Federal employees. Thus, the government as an employer assumes a direct interest in, and responsibility for, the health care of its career servants.

Moreover, the government may be expected to scrutinize the effectiveness of the coverage provided in order to assess the capacity of voluntary programs to function in an acceptable fashion in meeting the public's need for "prepaid" health services. Thus, the voluntary system of prepayment may be said to be on trial.

Southwestern Surgical Congress To Meet in Las Vegas

Site of the Twelfth Annual Meeting of the Southwestern Surgical Congress will be the Riviera Hotel, Las Vegas, Nevada, March 28, 29, 30, 31, 1960.

Highlighting the four-day meeting will be the appearance of five guest speakers whose talks will be included in over sixty widely diversified papers which will be presented. General discussion will be encouraged following the papers.

Guest speakers will be: Rutherford S. Gilfillan, M.D., Assistant Clinical Professor of Surgery, Stanford University, San Francisco; Walter C. Graham, M.D., Attending Surgeon, St. Francis Hospitals, Santa Barbara General Hospital, Santa Barbara, California; Bernard Hanley, M.D., Clinical Professor, University of Southern California, Los Angeles; Wm. P. Longmire, Jr., M.D., Professor of Surgery and Chairman, Department of Surgery, School of Medicine, University of California, Los Angeles; and Ian G. MacDonald, M.D., Clinical Professor, University of Southern California, Los Angeles.

Blocks of tickets for all major shows in Las Vegas have been reserved for persons attending the meeting. Arrangements have also been made for golf, boating, fishing and a side trip to Boulder Dam.

Special entertainment includes a cocktail party and the President's Banquet. A luncheon and bingo party have been planned for the ladies.

Registration for non-members will be $10.00. There will be no registration fee for members of the Southwestern Surgical Congress, Southeastern Surgical Congress, medical personnel, wives and program participants.

Oklahoma Dietetic Association Holds Annual Meeting

The twenty-second Annual Meeting of the Oklahoma Dietetic Association was held March 11 and 12 in Oklahoma City.

Guest speakers and their topics were: Sylvia D. Richardson, M.D., Assistant Professor of Pediatrics and Director of the Child Study Center, University of Oklahoma Medical Center, "Phenylketonuria in Oklahoma —A Preliminary Survey"; Jeremiah Stamler, M.D., Director of the Heart Disease Control Program, Chicago Board of Health, "Rationale and Methodology of a Coronary Prevention Evaluation Program;" and Rheba H. Edwards, M.D., Acting Director of the Oklahoma State Department of Mental Health, "The White House Conferences— Children and Youth, 1960, and Aging, 1961."

Additional topics discussed were selling, catering and disaster planning.

Those attending enjoyed over fifty exhibits on food and food service equipment.

Jefferson County Society Answers Forand

A program of free health care for persons who furnish proper certification of indigency is provided in a resolution recently approved by the Jefferson County Medical Society.

H. A. Rosier, M.D., spokesman for the society, reported that although physicians have traditionally offered free medical care to indigents, the trend toward socialized medicine (particularly the Forand Bill) inspired the formal resolution, which simply re-emphasizes the willingness of the society to continue the same policy.

The society has also gone on record regarding immunizations and office-dispensed antibiotics such as penicillin. Any person qualifying for assistance may obtain immunizations from the doctor or the county health nurse, and penicillin from the county health nurse.

Here is the resolution:

"Whereas the provision of health care to those unable to pay is primarily a community responsibility; and

Whereas this responsibility is not being met in some instances because of inadequate financing on the part of the county, the state and other welfare agencies; therefore be it

Resolved, that the members of the Jefferson County Medical Society will provide such care, after proper certification of those seeking assistance, by themselves, their pastor, their welfare worker or their county commissioner, that they are unable to pay for medical care and that no other funds from the county, state or other welfare programs are available to them."

Notice of the resolution was placed in newspapers of the county.

Exams Announced by American Board of OB-GYN

Applications for certification in the American Board of Obstetrics and Gynecology, new and reopened, Part I, and requests for re-examination in Part II are now being accepted. Deadline for receipt of applications is August 1, 1960.

It has been announced that after July 1, 1962, this Board will require a minimum of three years of approved progressive residency training to fulfill the requirements for admission to examination. After the above date, training by preceptorship will no longer be acceptable. Therefore, the initiation of preceptorships will not be approved after July 1, 1960.

Applications may be made to Robert L. Faulkner, M.D., 2105 Adelbert Road, Cleveland 6, Ohio.

IN ERROR

In the February, 1960 issue, *The Journal* erroneously stated that the new $800,000 University of Oklahoma Medical Research Building, now under construction, was an expansion of the Oklahoma Medical Research Foundation. We were in error, as the new structure is an addition to the Medical School and has no connection with the Research Foundation.

Doctor-Lawyer Banquet Held in Ardmore

Forty-five physicians and attorneys from Carter and adjacent counties attended the Fourth Annual Doctor and Lawyers Banquet to hear Jarrall E. Miller, M.D., Dallas radiologist and member of the Board of the Texas Medical Association, present and discuss many points of medico-legal interest. The speaker, in covering medical history and advances, gave great credit to the engineering profession and their efforts in promoting various sanitary measures.

According to Doctor Miller, radiology represents one of the ten world's greatest discoveries and has greatly assisted in the development of many other specialties. He pointed out that advancements are made by men having "honest and inquiring minds and with the courage to act." Therefore, he cautioned judges and lawyers, that although all modalities have some inherent dangers, and safeguards are necessary, they should not allow the potential dangers to outweigh the potential benefits. For example, in any area where a few hundred persons might presumably be harmed by the use of X-rays, the prohibition of its use could only mean unnecessary deaths for tens of thousands of people. Thus safeguards should not so strangle initiative that the use of various medical advances cannot be used and applied for the greatest good of the greatest number.

In closing the speaker stated: "Man does not possess truth; he acknowledges it. Medicine and law are devoted to truth. Both must show devotion; both must be persistent; both must seek the truth regardless of consequences; both must be more interested in truth than financial reward; both must take orders from a higher power than popularity, fame, or worldly success."

The 10 per cent of American families with the highest health expenditures in 1957-58 contributed almost 41 per cent of the total amount of expenditures by all families, according to a Health Information Foundation study. The 50 per cent of all families with the highest expenditures accounted for 88 per cent of the total outlay.



William T. Hawn, M.D., Binger physician, pictured left above, is shown receiving congratulations from Edward T. Cook, Jr., M.D. Others pictured, left to right, are Jerry Helms, Margaret Ann West, and Mrs. Lucille Freeman.

William T. Hawn, M.D. Is Honored

William T. Hawn, M.D., Binger physician for over fifty years, was honored January 31, 1960, at a joint celebration, when the Oklahoma State Medical Association presented him with a Fifty-Year Pin in recognition of his years of service to the medical profession. On the same occasion, Doctor and Mrs. Hawn were celebrating their golden wedding anniversary.

Presentation of the pin was made by Edward T. Cook, Jr., M.D., Anadarko physician.

The Binger doctor is a past president, vice-president and member of the Board of Directors of the Caddo County Medical Society. He is a Life Member of both his county medical society and the Oklahoma State Medical Association.

In addition to his private practice, Doctor Hawn has been active in both civic and church work, having served as mayor of Binger.

PHYSICIAN WANTED

Canton, Oklahoma needs a young, general practitioner to occupy new, community-owned clinic. Contact Earl Goode, Canton, Oklahoma.



Doctors Newell and Hamble Are Honored

Over forty members and guests of the Garfield-Kingfisher County Medical Society met at the Oakwood Country Club, Enid, February 24th to honor Waldo E. Newell, Sr., M.D., (left) and Virgil R. Hamble, M.D., (center) both of Enid, for having completed over fifty years in the practice of medicine. Walter E. Brown, M.D., (right) of Tulsa, President-Elect of the Oklahoma State Medical Association, presented the Association's 50-Year Pin awards to Doctors Newell and Hamble.

High Hopes for Tax Relief

Physicians and other self-employed have reason for optimism for tax relief. Specifically, the long-awaited Smathers-Morton-Keogh-Simpson Bill (H.R. 10) will likely receive favorable Congressional action this year, after nine years' promotion.

Last year, the House of Representatives passed the measure and referred it to the Senate Finance Committee where it rested without action until adjournment. It is again before the Finance Committee, which will have great influence on its final disposition.

Tax Equity for the Self-Employed

The purpose of the bill is to permit a self-employed person to defer income tax each year on $2500 or 10% of income, whichever is less, but not to exceed $50,000 during a lifetime. For those who have reached the age of fifty prior to the effective date, additional amounts may be set aside.

Kerr Responds to Telegram

The OSMA and fifteen other organizations of the self-employed (representing 115,000 self-employed Oklahomans) recently wired Senator Robert S. Kerr of their support for this legislation and requested his help as the second-ranking member of the Finance Committee.

In response to the telegram, Senator Kerr is now conducting a survey of the self-employed in Oklahoma, presumably to strengthen his position as a supporter of the bill.

In the meantime, the OSMA's Federal Legislative Committee has asked Oklahoma Physicians to write their senators and to send copies of their correspondence to Senator Harry Byrd, Chairman, Senate Finance, Committee, Senate Office Building, Washington, D.C.

Coming Meetings

UNIVERSITY OF OKLAHOMA MEDICAL CENTER
POSTGRADUATE PROGRAM*
Individual Postgraduate Courses

ADRENAL STEROIDS—March 24, 25, and 26. (Third Oklahoma Colloquy on advances in Medicine.)

ORTHOPEDIC SYMPOSIUM—May 13 and 14.

CARCINOMA OF THE SKIN—April 22 and 23.
ATHLETIC INJURIES

OKLAHOMA ASSSOCIATION OF HOUSE STAFF PHYSICIANS—May 6.

PEDIATRIC NEUROLOGY—May 12 and 13. This symposium to be held in conjunction with the Wednesday Short Course on May 11.

CARCINOMA OF THE SKIN—May 27-28. Surgery, Radiology, Pathology Conference.

SERIAL POSTGRADUATE COURSE
Postgraduate Division

Oklahoma City, Oklahoma
1959-1960

Wednesday Short Courses
3:30 to 8:30 p.m.

Apr. 13	Anesthesia for the Part-time Anesthetist
May 11	Neurological Diseases in Childhood
June 8	Practical Considerations of Neurological Problems

*Further information may be obtained from the Office of Postgraduate Education, University of Oklahoma School of Medicine, 801 N.E. 13th Street, Oklahoma City, Oklahoma.

OKLAHOMA CHAPTER
AMERICAN ACADEMY OF GENERAL PRACTICE

February 15-16, 1960 **Tulsa Hotel**
Tulsa, Oklahoma

The 12th Annual Meeting of the Oklahoma Chapter of the American Academy of General Practice will be held at the Tulsa Hotel, February 15-16, 1960, in Tulsa. Complete program on page 43.

21st Annual Meeting
SOUTHWEST ALLERGY FORUM

April 2, 3, 4, 5, 1960 **Oklahoma City**
Skirvin Hotel

The 21st Annual Meeting of the Southwest Allergy Forum will be held at the Skirvin Hotel, Oklahoma City, April 2, 3, 4, 5, 1960. Details of the program are outlined in the News Section of this issue of **The Journal.**

University of Texas
Postgraduate School of Medicine
MEDICAL SEMINAR

March 27, 1960 **Lake Texarkana**
Kickapoo Lodge

Lake Texarkana Cancer Seminar, the first of a series of medical seminars to be held at Kickapoo Lodge on Lake Texarkana, will be sponsored by the University of Texas Postgraduate School of Medicine, March 27, 1960. The program will run from 9:00 a.m. until 1:00 p.m.

Inquiries should be directed to William B. Harrell, M.D., Daniel-Harrell Clinic, Medical Arts Building, 317 State Line Avenue, Texarkana, Arkansas-Texas.

SOUTHWESTERN SURGICAL CONGRESS

March 28, 29, 30, 31, 1960 **Las Vegas, Nevada**
Riviera Hotel

The Twelfth Annual Meeting of the Southwestern Surgical Congress will be held at the Riviera Hotel, Las Vegas, Nevada, March 28, 29, 30, 31, 1960. Further details may be found in the News Section of this issue of **The Journal.**

SOUTHWESTERN SOCIETY FOR NUCLEAR MEDICINE

March 26-27, 1960 **Menger Hotel**
San Antonio, Texas

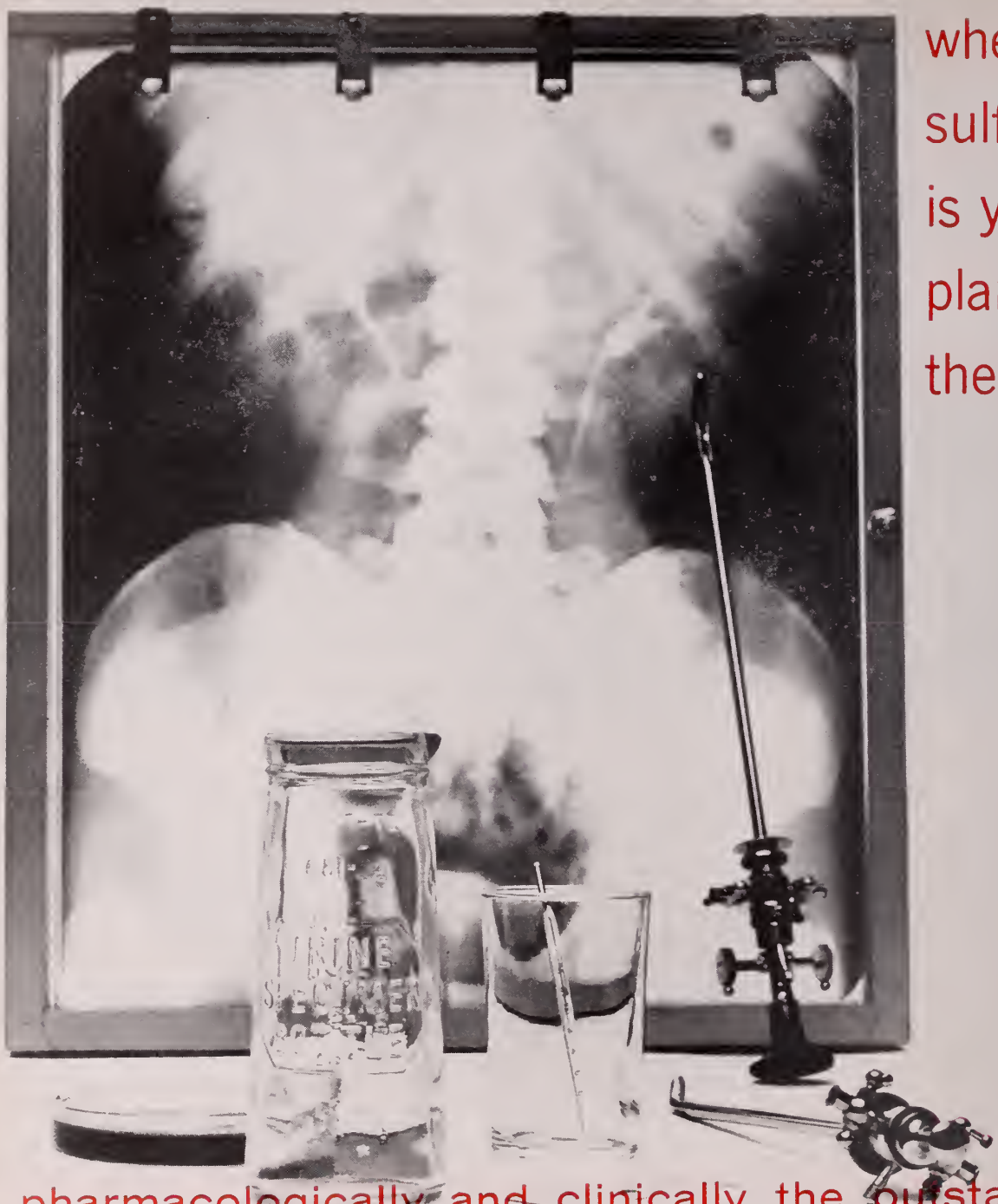
The Southwestern Society for Nuclear Medicine will meet March 26, 27, 1960 at the Menger Hotel in San Antonio, Texas. Details available from N. T. Werthessen, M.D., Program Chairman, Southwest Foundation for Research and Education, Rural Route 4, Box 86, San Antonio 7, Texas.

PAN-PACIFIC SURGICAL ASSOCIATION
EIGHTH CONGRESS

September 27-October 5, 1960 **Honolulu, Hawaii**

The Eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, Hawaii, September 27 through October 5, 1960. All members of the profession are eligible to register.

Further information and brochures may be obtained by writing to F. J. Pinkerton, M.D., Director General of the Pan-Pacific Surgical Association, Suite 230, Alexander Young Building, Honolulu 13, Hawaii.



when sulfa is your plan of therapy.

pharmacologically and clinically the outstanding

Rapid peak attainment — for early control — KYNEX® Sulfamethoxypyridazine reaches peak plasma levels in 1 to 2 hours[1,2] . . . or approximately one-half the time of other once-a-day sulfas.[2] Uninterrupted control is then sustained over 24 hours with the single daily dose . . . through slow excretion without renal alteration.

High free levels — for dependable control — More efficient absorption delivers a higher percentage of sulfamethoxypyridazine — averaging 20 per cent greater at respective peaks than glucuronide-conversion sulfas.[2] Of the total circulating levels, 95 per cent remains in the fully active, unconjugated form even after 24 hours.[3]

Extremely low toxicity[4] . . . only 2.7 per cent incidence in recommended dosage — Typical of KYNEX relative safety, toxicity studies[5] in 223 patients showed TOTAL side effects (both subjective and objective) in only six cases, all temporary and rapidly reversed. Another evaluation[4] in 110 patients confirmed the near-absence of reactions when given at the recommended dosage. High solubility of both free and conjugated product[6] obviates renal complications. No crystalluria has been reported.

Successful against these organisms: streptococci, staphylococci, E. coli, A. aerogenes, paracolon bacillus, Gram-negative rods, pneumococci, diphtheroids, Gram-positive cocci and others.

1. Boger, W. P.; Strickland, C. S., and Gylfe, J. M.: Antibiotic Med. & Clin. Ther. 3:378, (Nov.) 1956. 2. Boger, W. P.: Antibiotics Annual 1958-1959, New York, Medical Encyclopedia, Inc., 1959, p. 48. 3. Sheth, U. K.; Kulkarni, B. S., and Kamath, P. G.: Antibiotic Med. & Clin. Ther. 5:604 (Oct.) 1958. 4. Vinnicombe, J.: Ibid. 5:474 (July) 1958. 5. Anderson, P. C., and Wissinger, H. A.: U. S. Armed Forces M. J. 10:1051 (Sept.) 1959. 6. Roepke, R. R.; Maren, T. H., and Mayer, E.: Ann. New York Acad. Sc. 60:457 (Oct.) 1957.

KYNEX is your drug of choice



once-a-day sulfa . . .

OTE: Investigators note a tendency of some patients to sinterpret dosage instructions and take KYNEX on the miliar q.i.d. schedule. Since one KYNEX tablet is equivant to eight to twelve tablets of other sulfas, even modate overdosage may produce side effects. Thus, the ngle dose schedule must be stressed to the patient.

YNEX Tablets, 0.5 Gm., bottles of 24 and 100. Dosage: dults, 0.5 Gm. (1 tablet) daily, following an initial first y dose of 1 Gm. (2 tablets).

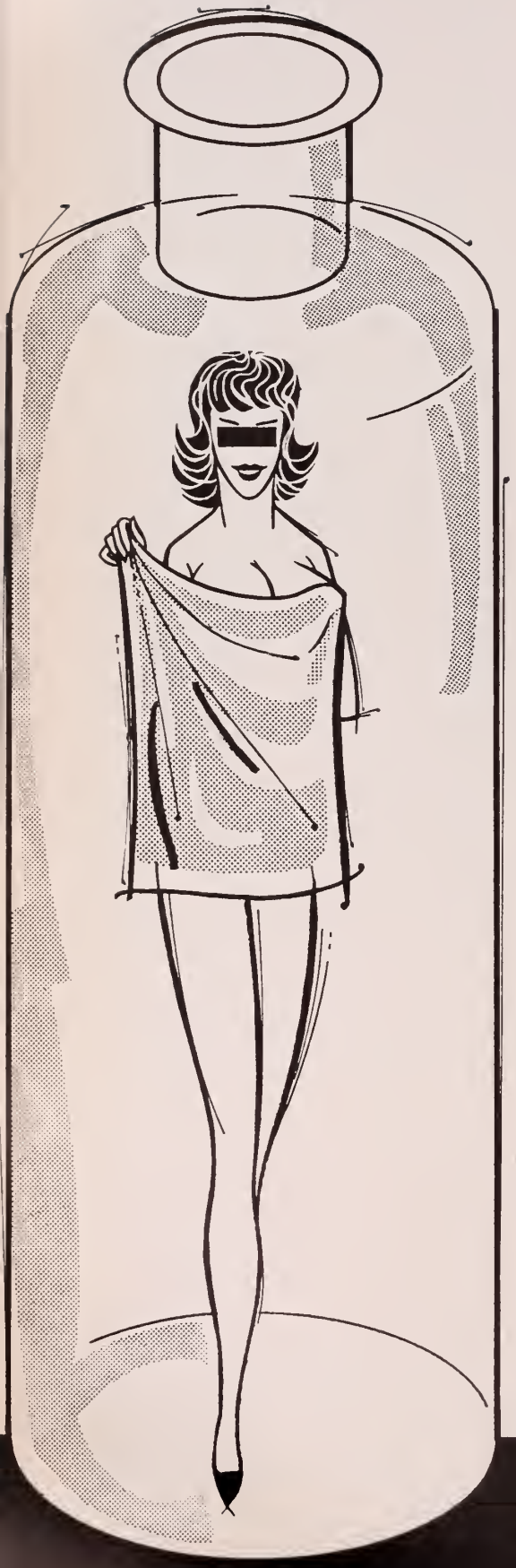
YNEX Acetyl Pediatric Suspension, cherry-flavored, 250 g. sulfamethoxypyridazine activity per teaspoonful (5 cc.). ottles of 4 and 16 fl. oz. Recommended Dosage: Children nder 80 lbs.: 1 teaspoonful (250 mg.) for each 20 lb. body eight, the first day, and ½ teaspoonful per 20 lb. per day ereafter. For children 80 lbs. and over: 4 teaspoonfuls .0 Gm.) initially and 2 teaspoonfuls daily thereafter. Give mediately after a meal.

KYNEX®

Sulfamethoxypyridazine Lederle

NEW—for acute G.U. infection AZO-KYNEX® Phenylazodiaminopyridine HCl—Sulfamethoxypyridazine Tablets, contains 125 mg. KYNEX in the shell with 150 mg. phenylazodiaminopyridine HCl in the core. Dosage: 2 tablets q.i.d. the first day; 1 tablet q.i.d. thereafter.

EDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York Lederle



Have something to exhibit

If so, you should apply now for space at the 1960 meeting of your association. Scientific Exhibits will again play a major role . . . so why not prepare a graphic presentation of your work and share it with the 700 physicians who will attend the state's largest medical meeting? A research or clinical project may be shown as an individual exhibit, or you may present a single case in the "Klinical Kaleidoscope" section.

Research or clinical project

Perhaps you have done some original research, undertaken some experimental work, or simply drawn some conclusions based upon a series of interesting cases. Then you would be eligible to apply for an exhibit booth of your own in the Scientific Exhibit Section. A subject which has a special visual interest would be particularly suitable . . . and it need not be large—just stimulating!

Bizarre case

All of us have encountered a unique case which is worth talking about. If you have a well-documented one which you would like to share with others, toss your hat in the ring today by completing the application on the opposite page. The "Klinical Kaleidoscope" section will be a varied selection of bizarre cases, each presented with one to five x-rays or illustrations and accompanied by a very brief abstract of the clinical course.

O.S.M.A. ANNUAL MEETIN

municipal auditorium · oklahoma ci

may 2·3·4, 1960

APPLICATION FOR SCIENTIFIC EXHIBIT SPACE

54th Annual Meeting

OKLAHOMA STATE MEDICAL ASSOCIATION

Municipal Auditorium — Oklahoma City — May 2, 3, 4, 1960

1. NAME AND ADDRESS OF APPLICANT:

2. TITLE OF EXHIBIT:

3. INDIVIDUAL BOOTH? ☐ "KLINICAL KALEIDOSCOPE" SECTION? ☐

4. SPACE REQUIRED:

   A. BOOTH: Indicate width in multiples of eight feet _____

   (All booths will be 6 feet in depth)

   B. "KLINICAL KALEIDOSCOPE": Indicate square footage:

   Wall-type display _____ Table top _____

5. A. GENERAL DESCRIPTION:

   B. MATERIAL TO BE SHOWN (Subject matter):

   C. METHOD OF PRESENTATION (x-ray, photos, graphs, models, etc.):

6. PREVIOUS SHOWINGS

ACT NOW — SPACE IS LIMITED

Applications Must Be Received Prior to March 1, 1960

Oklahoma State Medical Association • Box 9696 • Oklahoma City

Auxiliary News

Planning Time

Planning toward any important event involves the date, the time, the place, and the guests to be invited. If the event has an important reason for being held, so much the better. Our current interest contains all of these elements—Membership. In charge are our two Vice-Presidents of State and our District Councilors. Hostesses should include executive board members and every local woman interested in our Auxiliary. The time: Now. The place: Our country.

Guest List

Our guest list is obvious—every eligible physician's wife. In some communities, therefore, the list is small and of necessity, exclusive. In others, the list might be so large as to be over-whelming. We might be tempted not to send an invitation, or to have little concern if no interest is shown. As in ordinary social courtesy, regretted invitations usually contain the reason for the regret. It is of great importance to know in advance when a year's work in Auxiliary is being planned. How many memberships? How many participants can be expected?

Membership as a Challenge

Here our "party" analogy must end. Because in personal affairs we cannot sell the deliner, we might extend this attitude toward membership in the Auxiliary. This cannot be done. Mrs. Clifford M. Bassett, our State President, quoted Henry Ford in her program at Fall Conference in Chicago: "Meeting together is a beginning. Staying together is success. And working together is progress."

Obviously, the first point, meeting together, can be accomplished only if interest in the prospective member is first created. Contacts of personal nature are invaluable when a doctor's wife has not had an Auxiliary affiliation. Invite her to meetings, not on an "I'll see you there" basis. Invite her to go with you; this is important. Few women will go alone to a strange home or meeting-place to meet a group of unfamiliar people. Through orientation with Auxiliary functions, contacts with members, and familiarity with its projects, her interest and her desire to join will logically follow.

What about those members who have been eligible for some time, but disinterested? Or those who have dropped out? Mrs. Mark Yessian of Rhode Island submitted the following ideas: " . . . Who is the dropped-out member? The president writes a personal letter saying how very sorry she is that the member is not now active and hopes to see her at the next meeting. Why has she dropped out? Strangeness in the group? The hospitality chairman (membership chairman or councilor) makes a special effort to see that the member is either escorted or met at the next meeting. Are her interests elsewhere? Fine, but we, too, can use her interests to the benefit of the auxiliary activities. The program chairman can incorporate some of her interests in her planning for the coming year or make certain that the particular talent of this member is made available to one of our committees. She is needed and wanted. Is it physically impossible to attend a meeting or participate? Passive membership is then encouraged with the idea imparted that numerical strength is also needed if we are to keep abreast of all the involved issues that affect our husbands and their practices."

Further breakdown on the "physically impossible to attend" statement, or just the excuse "impossible to attend," leads to many loop-holes. Many young mothers use P.T.A., church work, varied children's activities, as reasons for not being active. Some should-be members use as an excuse a preference for other organizations.

Second-Bests

To an eligible member who has preceding attitudes, Mrs. Remer Y. Clark of Georgia responds, "I did not know there were any doctors' wives who preferred other organizations. I just bet there aren't. Why should

a woman prefer second best when she can belong to the auxiliary to the highest profession in the world?" To the case of the young mother who simply does not have time for the medical auxiliary, Mrs. Clark has this answer: "Being a mother is so important. But did she ever stop to think that she was a wife first? Her first allegiance is to her husband. Besides, isn't she embarrassed in PTA admitting she knows nothing of medical policy—being on the board without knowing what to say when the chiropractor was invited to speak at a meeting. Or worse still, just looking real blank when it was suggested that they invite Mr. Forand for a few words. . . ."

Exaggerated? Not really. Too often the non-member may have a years-old interpretation of the auxiliary. Things have changed; we are constantly on the alert to present new ideas in programming, to make the process of "education" a happy experience to those who are averse to the idea of learning, instead of being entertained. An old grievance? Don't laugh. Some former members or "drop-outs" may rely on this excuse year after year, complaining of unfriendliness, lack of interest from the auxiliary in her own pet project—any number of personal reasons. Persistence in these cases might pay surprising dividends. A visit in these cases might overcome the first objection; a few years and perhaps added maturity may cause her to see the childishness of the second attitude. In any event, she should be given the opportunity of becoming an active member once more.

Results

In conclusion, we should examine the results of concentrated, concerted efforts in membership. Increase in numbers? Of course, any auxiliary is proud of an increase in members, particularly if the new members are active, alert and interested. With numbers, as one member states, we present an organized front. We cannot advertise our projects, on the whole, as most service groups do. And since our projects do not command columns of publicity for our charities and volunteer work, posters and billboards, some doctors' wives automatically minimize their importance.

As an organized front, with the intellectual and social capacities of a united membership, we can face problems of medical legislation, community health, whatever we must. Convince yourself; convince every wife of every doctor. It is that simple.

25 YEARS AGO



Articles published in *The Journal* of the Oklahoma State Medical Association, March, 1935.

Editorial

THE MEDICAL SCHOOL

It seems a shame that the Medical School of our University has to be made a target by politicians. Why is it that a so well conducted institution of higher learning cannot be managed by its Dean, the President of the University and Board of Regents? This is a perfectly legal procedure and satisfactory to everyone unless he may have some political axe to grind. The people of Oklahoma in general, and the medical profession in particular, have watched with pride the development of our Medical School from a Class C institution to Class A, recognized throughout the nation as presenting a teaching curriculum of high standards and graduating into the medical profession men and women well fitted to practice the healing art. We have been particularly fortunate in having the services of such men as Drs. LeRoy Long and L. J. Moorman to head this school, and while they may not be educated to cope with the politician when he attempts to create false impressions as to the conduct of the institution, they have both worked faithfully and efficiently in the development of a fine medical school and should not be interfered with by politicians but left as they should be, responsible to the President of the University and the Board of Regents.

If the politicians will keep their hands off and leave the conduct of these institutions to trained educators this high standard will be retained. Conversely, if the medical department is booted about by investigating committees composed of men not trained in medical education, it will become a political institution, lose its classification and the former students can only blush when reference is made to their alma mater.

May the people of the state see to it that political manipulation does not destroy the fine work and cooperative spirit existing in the faculty and keep the administration of our Medical Department in the hands of those trained to manage such an institution.

Deaths

McLain Rogers, M.D.
1874-1960

McLain Rogers, M.D., Clinton physician, civic leader and Past-President of the Oklahoma State Medical Association, died in Clinton, February 5, 1960.

Born in Clyde, North Carolina in 1874, Doctor Rogers graduated from Emory University, Atlanta, Georgia in 1902. After practicing seven years in Geary, he came to Clinton in 1909, later establishing Clinton's first hospital.

Doctor Rogers was active in assembling data for the State Legislature when the Basic Science Law was passed. For his work in this field, he was presented an honorary-life membership in the Oklahoma State Medical Association in 1954.

He served as President of the OSMA in 1922-23 and was listed in "Who's Who" in the 1920s.

For 12 years, he served as a member of the state association's Public Policy Committee. In 1958, he was honored when the OSMA presented him with a Fifty Year Pin in recognition of his years of service to the profession.

Active in many branches of civic work, Doctor Rogers served as mayor of Clinton for 23 years.

He held a Life Membership in the Boy Scouts of America, was a member of the Southern Surgical Congress, the Clinical Congress of Surgeons, the Oklahoma Congress of Parents and Teachers and was a Fellow in the American College of Surgeons.

James David Markland, M.D.
1894-1960

James David Markland, M.D., Tulsa physician and allergist, died January 25, 1960 in Tulsa.

Born in Kokomo, Indiana in 1894, Doctor Markland moved to Oklahoma in 1907, settling in Altus. He graduated from the University of Tennessee College of Medicine in 1936.

Past president of the Tulsa Internist Society, Doctor Markland was also a member of the Southwest Allergy Society and former member of the board of directors of Hillcrest Medical Center.

Books Received

The following books have been received by *The Journal* office. As space permits and the context warrants, books will be reviewed.

The Acute Medical Syndromes and Emergencies. Albert S. Hyman, M.D., Landsberger Medical Books, Inc., New York. Price $8.75.

Babies By Choice or By Chance. Alan F. Guttmacher, M.D., Doubleday & Company, New York. Price $3.95.

The Care of Minor Hand Injuries. Adrian E. Flatt, M.D., C. V. Mosby Co., St. Louis, Mo. Price $9.50.

The Cigarette Habit: A Scientific Cure. Arthur King, Doubleday & Co., Inc., New York. Price $2.00.

A Cookbook for Diabetics. From the ADA Forecast by Deaconess Maude Behrman, American Diabetes Association, New York.

The Emergency Syndromes in Pediatric Practice. Alfred J. Vignec, M.D., Landsberger Medical Books, Inc., New York. Price $9.00.

Family Medical Encyclopedia. Justus J. Schifferes, Ph. D., Permabooks, New York. Price $.50.

Guide to Better Health. Harry J. Johnson, M.D., Prentice-Hall, Inc., Englewood Cliffs, New Jersey. Price $4.95.

Jewish Medical Ethics. Immanuel Jakobovits, Philosophical Library, New York. Price $6.00.

Living Beyond Your Heart Attack. Eugene B. Mozes, M.D., Prentice Hall, Inc., New York. Price $3.50.

Master Your Tensions and Enjoy Living Again. George S. Stevenson, M.D. and Harry Milt, Prentice Hall, Inc., Englewood Cliffs, New Jersey. Price $4.95

Medieval and Renaissance Medicine. Benjamin L. Gordon, Philosophical Library, Inc., New York. Price $10.00.

The Modern Family Health Guide. Morris Fishbein, M.D., Doubleday & Company, Inc.. Garden City, New York. Price $7.50.

PHYSICIAN PLACEMENT

Dermatology

Gordon Harold Ekblad, M.D., Field Medical Service School, Camp Pendleton, California, 53, married, University of Minnesota, 1930, available January 1960.

General Practice

Johnny Bill Delashaw, U.S.P.H.S. Hospital, Wyman Park Drive and 31st Street, Baltimore 11, Maryland, age 27, married, University of Texas Medical Branch, 1959, available July 1960.

Robert Goodman, M.D., 63 Old Gate Lane, Powers Lake, North Dakota; age 56; married; University of Manitoba, 1930; no military obligations; available anytime 1960.

Richard Fred Harper, M.D., Conway Memorial Hospital, Monroe, Louisiana; age 28, married; Oklahoma University, 1956; available July 1960; prefer associate practice.

Sherman Allen Hope, M.D., 1105 Yale Ave., Panama City, Florida, PO 3-6656, age 27, married, University of Oklahoma School of Medicine, 1957, available July 1960.

Paul J. Vega, M.D., 6070 Orleans Ave., New Orleans, Louisiana, age 26, married, Louisiana State University, 1956, no military obligations, available now.

Thomas S. Whitecloud, M.D., 858 Market Street, Pascagoula, Mississippi, age 45, married, Tulane, 1943, veteran, has interest in teaching, availability date depends on situation.

Internal Medicine

Jack Brook, M.D., 315 Hatton Point Road, Portmouth, Virginia; age 32; married; New York Medical College, 1953; eligible Board of Internal Medicine; specialty, Internal Medicine & Hematology; available anytime.

Richard I. Hochman, M.D., 37, Badger Road, Annapolis, Maryland, 31, married, New York University, 1952, available October 1, 1960.

Clare Charles Hugan, Jr., M.D., 9121 Prest, Detroit 28, Michigan; Emory University 1953; Board requirements will be completed July 1960; available July 1960.

Joseph Chester Maternowski, M.D., 319 Highlake, Ann Arbor, Michigan; age 35; married; St. Louis University, 1953; eligible board of Internal Medicine & Allergy; veteran; specialty, Internal Medicine & Allergy; available July 1, 1960.

Van B. Saye, Jr., M.D., 1230 Brockenbraugh Court, Metairie, Louisiana, age 28, married, Medical College of Georgia, 1954, Air Force Reserve, available July 1, 1960.

Locum Tenens

Harold W. Calhoon, M.D., Wesley Hospital, Oklahoma City. One year residency in Internal Medicine. Available July 1 to September 15th, 1960.

Arnold Giesbrecht, M.D., Hallock, Minnesota, age 34, married, graduate Winnipeg, Manitoba, 1957, prefers suburban or locality close to cities.

James D. Green, M.D., second year resident in Internal Medicine at St. John's Hospital, Tulsa, desires two weeks' tenure between now and July 1. Write to St. John's or call RIverside 3-3059.

Russell M. Preston, M.D., 476 San Juan, Oak Harbor, Washington, desires six month's work in General Practice between time of separation from military service to beginning of residency training, January-July, 1960.

Obstetrics and Gynecology

Lynn W. Abshere, M.D., 708 N.E. 14th, Oklahoma City, age 31, married, University of Oklahoma School of Medicine, 1956, American College of OB-GYN, no military obligations, available July 1, 1960.

Edmond Michael Brophy, M.D., 1823 Portsmouth, Westchester Illinois; married; University of Illinois, 1946; Board eligible upon completion of practice requirements; no military obligations; available July, 1960.

Robert Russell Fahringer, M.D., P.O. Box 148, Danville, Pennsylvania; age 35; married; Jefferson Medical College of Philadelphia, 1953; inactive military status, USNR; prefers full or part-time teaching; available February 1960.

David H. Holmes, M.D., 1017 Grovena Drive, St. Louis, Missouri, 37, married, Washington University, St. Louis, Veteran, available July 1, 1960.

Gerald R. Keilson, M.D., Medical Arts Building, Dallas, Texas, age 31, married, University of Texas, 1953, board qualified, veteran, available in July 1960.

Nejdat Mulla, M.D., 1110 Belmont Avenue, Youngstown, Ohio, age 36, married, University of Geneva, Switzerland, 1952, American College of OB-GYN, not eligible military service, available July 1, 1960.

Harold G. Ray, M.D., 212 Darien Place, Box 503, Balboa Heights, Canal Zone, age 28, married, University Arkansas School of Medicine, 1955, now in active reserve, available July 1, 1960.

Harold E. Reid, M.D., 519 N. Lincoln St., Stanton, Michigan; age 42; married; University of Michigan, 1942; OB-GYN, also interested in industrial; available February 1960.

Alvon C. Winegar, M.D., 1940 Michigan, Benton Harbor, Michigan, age 41, married, Wayne University, 1942, no military obligation, available early 1960.

Pediatrics

H. M. McClintock, M.D., 5712 St. John St., Kansas City, Missouri, 29, married, Baylor University, 1955, veteran, available December 1960.

Orthopedics

Newsom Stool, M.D., c/o Mayo Clinic, Rochester, Minnesota; age 30; married: Graduate Baylor University, 1953; American Board of Orthopedic Surgery; no military obligations; available July 1960.

Radiology

Gerhard T. Schmunk, M.D., 3530 S. Glencoe, Denver 22, Colorado; age 36; married; University of Nebraska College of Medicine, 1952; Board eligible July 1, 1960; veteran; consider group or associate practice; available July 1, 1960.

Surgery

(Name on Request) 32 years old, married, Tulane, 1952, veteran, board eligible.

Clarence I. Britt, M.D., 1650 Neil Avenue, Apt 15, Columbus, Ohio; age 32; married; Ohio University College of Medicine, 1951; American Board of Surgery 1959; eligible American Board of Thoracic Surgery, 1960; inactive reserve; available July 1960.

Delbert H. McGinnis, M.D., Box 75, Clinton-Sherman AFB, Oklahoma; age 28; married; end military obligation July 3, 1960; interested in general practice; available July 4, 1960.

Sam Leslie Robinson, M.D., University Medical Center, Jackson, Mississippi; age 30; married; University of Tennessee, 1953; inactive reserve; available July 1961.

Olin O. Williams, Jr., M.D., 3768 Alma Drive, Memphis, Tennessee, 29, married, University of Tennessee, veteran, available January 1962.

Urology

Alex Grossman, M.D., V.A. Center, Temple, Texas, 42, married, University of Texas, 1951, board certified, veteran, available now.

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MISCELLANEOUS ADVERTISEMENTS

FOR SALE: Autoclave, Castle, electric. Originally purchased for $1866.75 in 1956. Contact Walt Allen, 116 North Fourth Street, Chicaksha, Oklahoma.

FOR SALE or RENT: Office building, equipment and supplies, including x-ray. Residence also available. Contact Box F, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: $40,000 per year General Practice. 14 room completely equipped, air conditioned clinic. County seat town of 4,000 and 20,000 trade territory with new 40 bed hospital. Small down payment and terms to suit buyer. Write Key J, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: General practice, established six years, in oil and ranching community. Town population is 3,000 with trade area over 15,000. Closest physician 30 miles. Netting $20,000 to $24,000 per year. Located in modern, leased clinic building. Presently in partnership but continued partnership not mandatory. 18-bed, modern, community hospital. Only two physicians in town. Reason for selling, physician returning to Hawaii. No down payment necessary. Contact William G. Mays, M.D., Fairfax, Oklahoma.

WANTED: Young General Practitioner, without military obligations, to become partner in established clinic and hospital practice. $1000.00 per month to start. Contact Neumann-Ottis Clinic, Okarche, Oklahoma.

DEPUTY FLIGHT SURGEON wanted for Federal Aviation Agency. The 40 hours-per-week position pays an annual salary of $12,555, plus Civil Service benefits including a retirement program, annual and sick leave policy, group life insurance and others. Physician must have completed the basic course in Aviation Medicine prescribed by the U.S. Military Service, and have three years professional experience in internal medicine. Write FAA, Region 2, Box 1689, Ft. Worth 1, Texas.

PHYSICIANS OFFICES available in new medical center, located on North Western in Oklahoma City. Approximately 500 square feet per unit. Ample parking. Now being constructed. Contact Bob Fowler, P.O. Box 9554, Shartel Station, Oklahoma City, WI 2-1021.

WANTED: Internist to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR RENT: Office space for two doctors, modern, air conditioned building. 3832 N. May Ave., Oklahoma City. Complete maintenance. Free parking space for patients. WI 2-5531.

WANTED: Surgeon to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

WANTED: General Practitioner with some ability to do surgery. Primarily industrial practice. Beginning salary $900.00. Contact C. J. Paternostro, M.D., 426 Medical Arts Building, Dallas, Texas.

FOR SALE: Practice, office building and equipment in Harlingen, Texas. 1,000 square feet of floor space, six rooms equipped for Eye, Ear, Nose and Throat work, small laboratory and dark room. Building valued at $15,821.00. Equipment $6,720. Contact Edward M. Loyd, M.D., P.O. Box 2061, Harlingen, Texas.

FOR SALE: General practice partnerships established 7 years in prosperous oil and agriculture community. Town of 5000 and trade area over 15,000 population. Gross receipts exceed $50,000 annually. New clinical building for sale or rent. Practice is priced reasonably and terms can be arranged. Owners going into residency. Contact Box L, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

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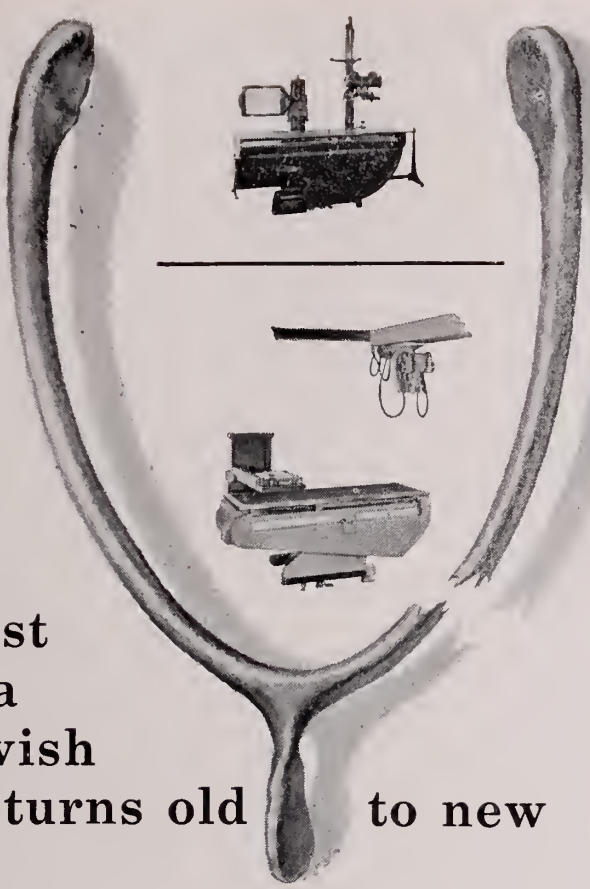


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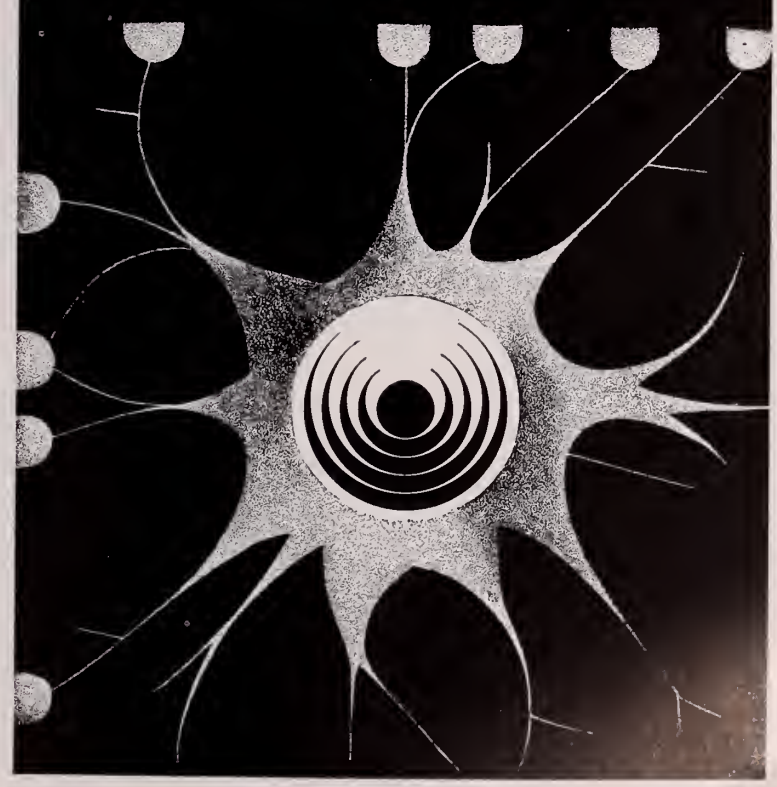
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References: 1. Ayd, F. J., Jr.: Bull. School Med., Univ. Maryland *44*:29, 1959. 2. Azima, H., and Vispo, R. H.: A.M.A. Arch. Neurol. & Psychiat. *81*:658, 1959. 3. Lehmann, H. E.; Cahn, C. H., and de Verteuil, R. L.: Canad. Psychiat. A. J. *3*:155, 1958. 4. Mann, A. M. and MacPherson, A. S.: Canad. Psychiat. A. J. *4*:38, 1959. 5. Sloane, R. B.; Habib, A., and Batt, U. E.: Canad. M.A.J. *80*:540, 1959. 6. Straker, M.: Canad. M.A.J. *80*:546, 1959. 7. Strauss, H.: New York J. Med. *59*:2906, 1959.

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Obviously, in any special diet, the fewer required changes in the patient's eating habits, the more likelihood there is that the patient will adhere to the prescribed diet.

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This flaky pie crust, *crisp cookies, Chiffon cakes, biscuits can all be made easily with Wesson. Decrease the calories of pie by preparing with single crust and a fresh fruit or gelatin filling. It is delicious.*

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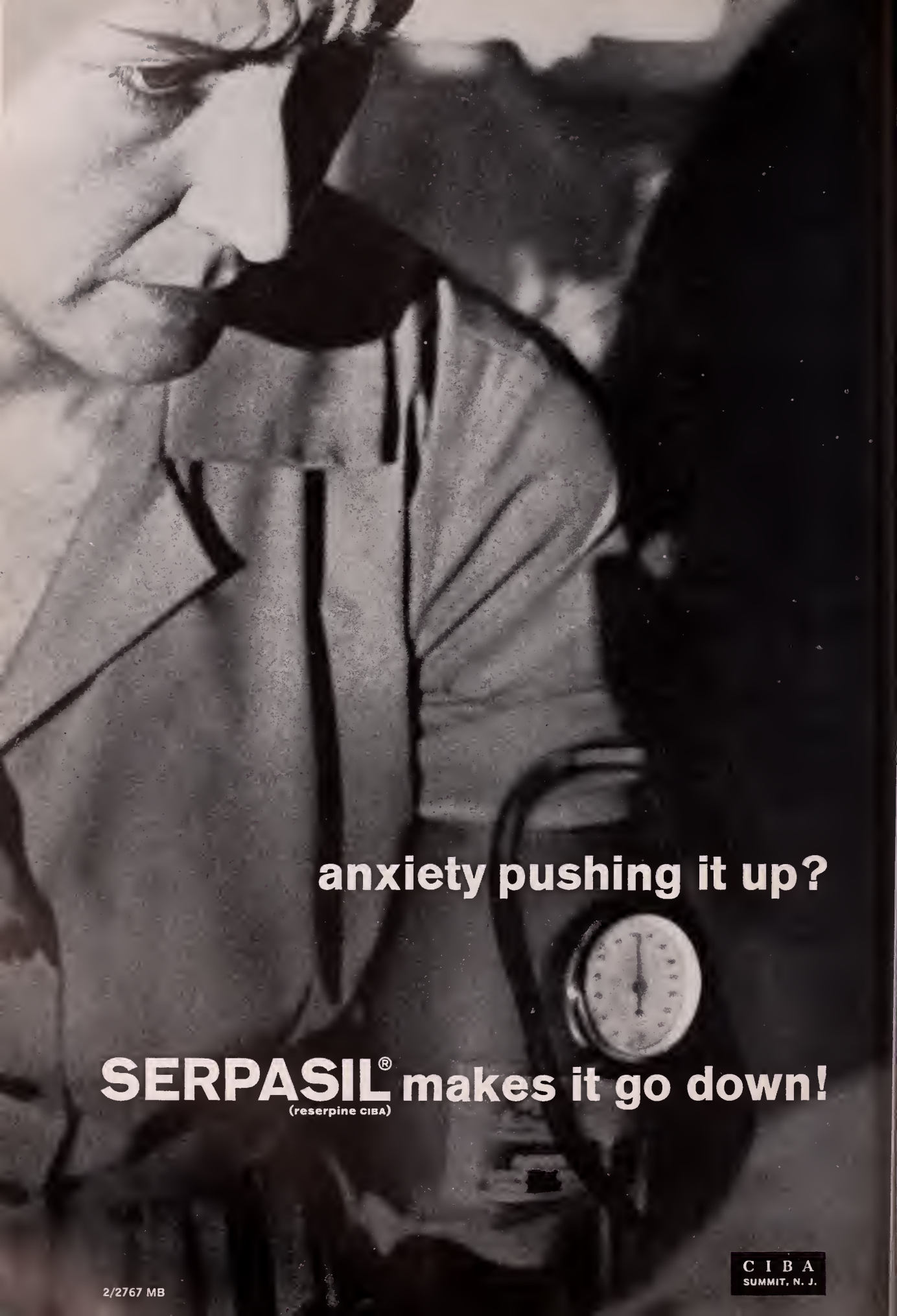
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Diagnostic Quandaries

Colitis? Gall Bladder Disease?
Chronic Appendicitis?
Rheumatoid Arthritis? Regional Enteritis?

A DISEASE that is frequently overlooked in solving diagnostic quandaries is amebiasis. Its symptoms are varied and contradictory, and diagnosis is extremely difficult. In one study, 56% of the cases would have been overlooked if the routine three stool specimens had been relied on.[1]

Another study found 96% of a group of 150 patients with rheumatoid arthritis were infected by *E. histolytica*. In 15 of these subjects, nine stool specimens were required to establish the diagnosis.[2]

Webster discovered amebic infection in 147 cases with prior diagnoses of spastic colon, psychoneurosis, gall bladder disease, nervous indigestion, chronic appendicitis, and other diseases. Duration of symptoms varied from one week to over 30 years. In some cases, it took as many as six stool specimens to establish the diagnosis of amebiasis.[3]

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Supplied in bottles of 40 tablets, each tablet containing 50 mg. of glaucarubin.

Write for descriptive literature, bibliography, and dosage schedules.

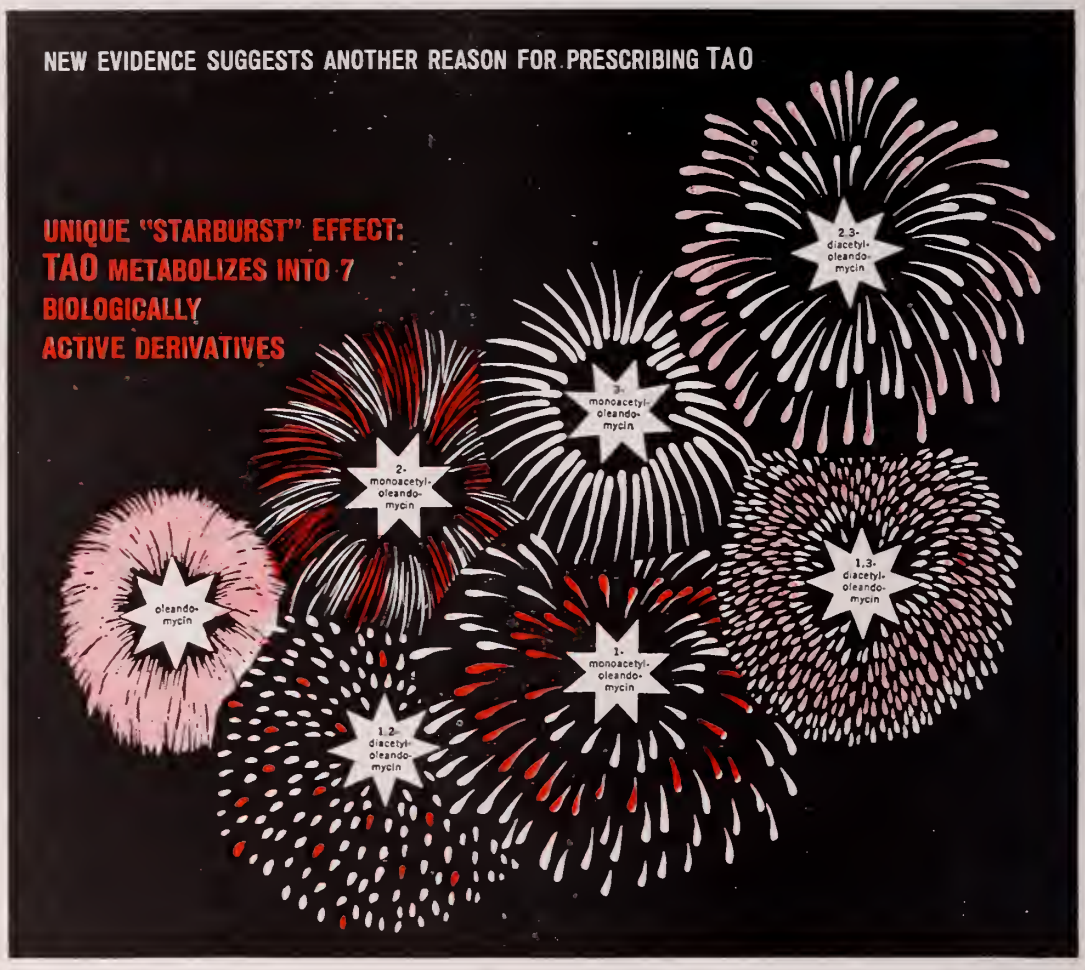
1. Cook, J.E., Briggs, G.W., and Hindley, F.W.: Chronic Amebiasis and the Need for a Diagnostic Profile. Am. Pract. and Dig. of Treat. *6*:1821 (Dec., 1955).

2. Rinehart, R.E., and Marcus, H.: Incidence of Amebiasis in Healthy Individuals, Clinic Patients and Those with Rheumatoid Arthritis, Northwest Med., *54*:708 (July, 1955).

3. Webster, B.H.: Amebiasis, a Disease of Multiple Manifestations, Am. Pract. and Dig. of Treat. *9*:897 (June, 1958).

*U.S. Pat. No. 2,864,745

THE S.E. MASSENGILL COMPANY
BRISTOL, TENNESSEE
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The impression that TAO is an unusually active antibiotic has steadily gained recognition by impressive clinical performance. Now come reports of **in vivo** and **in vitro** biological and biochemical evaluations that show TAO to be indeed unique.[1,2]

TAO differs from other antibiotics in that it is metabolized to multiple active compounds which **remain** active throughout their presence in the body. These 7 derivatives (in addition to TAO) show activity against common Gram-positive pathogens, including resistant strains of **Staph. aureus.**

In light of these findings, take another look at TAO performance: • 92% success in published cases of Gram-positive respiratory, skin, soft tissue and genitourinary infection • Effective against 78% of 64 "antibiotic-resistant" epidemic staphylococci. (In the same study, chloramphenicol was active against 52%; erythromycin against only 25%)[3] • No side effects in 94%; infrequent reactions mild and easily reversed • Quickly absorbed • Highly palatable.

Sound reasons to: Start with TAO to end 9 out of 10 common Gram-positive infections.

Supplied: TAO Capsules—250 mg., and 125 mg., bottles of 60. **TAO for Oral Suspension**—125 mg. per tsp. (5 cc.) when reconstituted; unusually palatable cherry flavor; 60 cc. bottle. Prescription only.

Other TAO forms available: TAO Pediatric Drops: flavorful, easy to administer. **TAO®-AC:** TAO analgesic, antihistaminic compound. **TAOMID®:** TAO with triple sulfas. **Intramuscular or Intravenous:** in clinical emergencies. Prescription only.

1. English, A. R., and McBride, T. J.: Proc. Soc. Exper. Biol. & Med. 100:880 (Apr.) 1959. 2. Celmer, W. D.: Antibiotics Annual 1958-1959, New York, Medical Encyclopedia, Inc., 1959, p. 277. 3. English, A. R., and Fink, F. C.: Antibiotics & Chemother. 8:420 (Aug.) 1958.

designed for superior control of common Gram-positive infections

Tao®

(triacetyloleandomycin)

Capsules/Oral Suspension



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Science for the World's Well-Being



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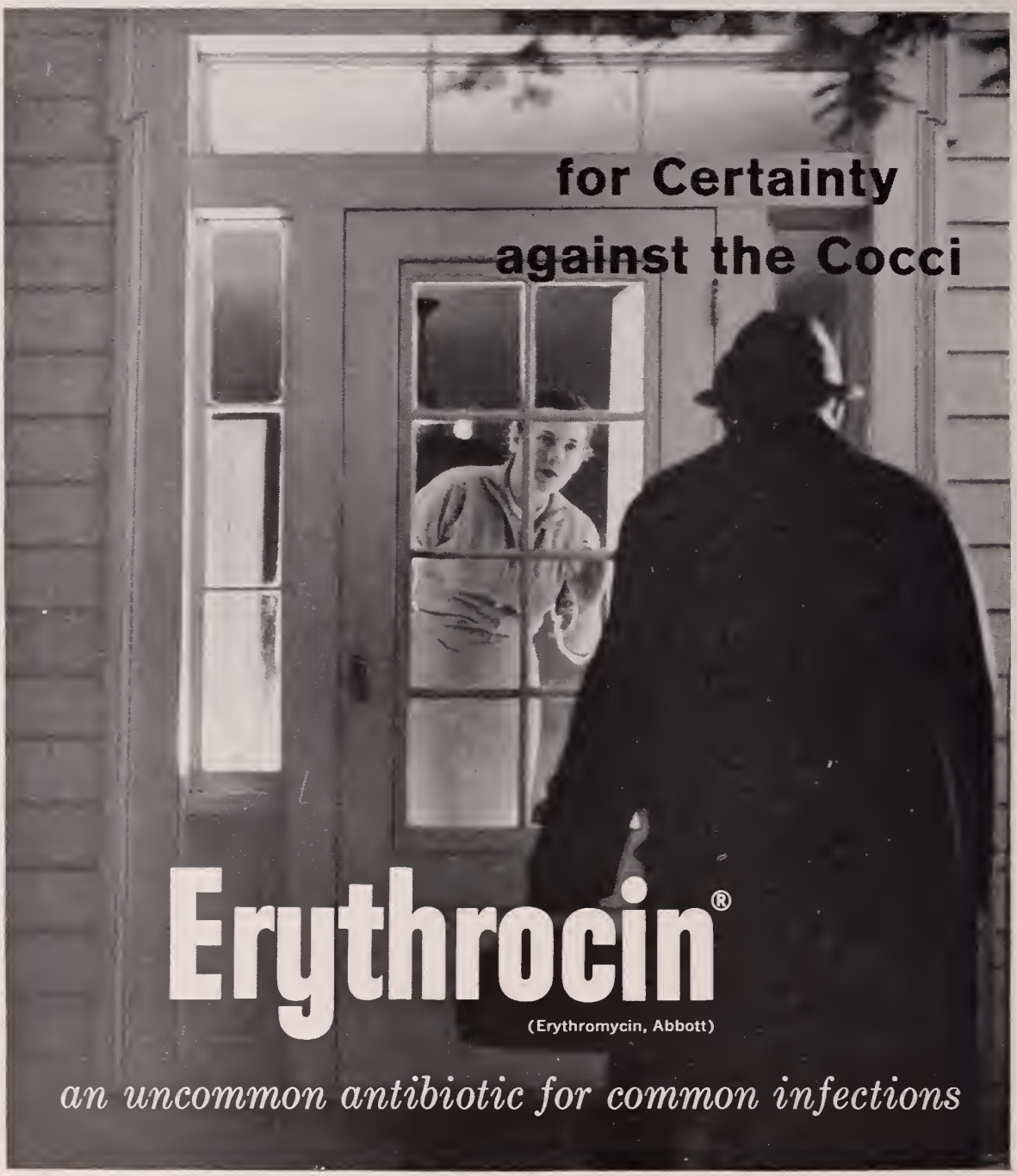
relieves pain,
muscle spasm,
nervous tension

rapid action • non-narcotic • economical

"We have found caffeine, used in combination with acetylsalicylic acid, acetophenetidin, and isobutylallylbarbituric acid, [Fiorinal] to be one of the most effective medicaments for the symptomatic treatment of headache due to tension."
Friedman, A. P., and Merritt, H. H.: J.A.M.A. *163*:1111 (Mar. 30) 1957.

Fiorinal – available as tablets and *capsules* (new form). *Each contains:* Sandoptal (Allylbarbituric Acid N.F. X) 50 mg. (¾ gr.), caffeine 40 mg. (⅔ gr.), acetylsalicylic acid 200 mg. (3 gr.), acetophenetidin 130 mg. (2 gr.).
Dosage: 1 or 2 every 4 hours, according to need, up to 6 per day.

SANDOZ



Provides fast, high blood and tissue concentrations—plus an unparalleled safety record. Erythrocin is available in easy-to-swallow Filmtabs® (100 and 250 mg.); in tasty, citrus-flavored Oral Suspension (200 mg. per 5-cc. teaspoonful); and for intravenous and intramuscular use.

ABBOTT

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Of course, women like "Premarin"®

THERAPY for the menopause syndrome should relieve not only the psychic instability attendant the condition, but the vasomotor instability of estrogen decline as well. Though they would have a hard time explaining it in such medical terms, this is the reason women like "Premarin."

The patient isn't alone in her devotion to this natural estrogen. Doctors, husbands, and family all like what it does for the patient, the wife, and the homemaker.

When, because of the menopause, the psyche needs nursing—"Premarin" nurses. When hot flushes need suppressing, "Premarin" suppresses. In short, when you want to treat the whole menopause, (and how else is it to be treated?), let your choice be "Premarin," a complete natural estrogen complex.

"Premarin," conjugated estrogens (equine), is available as tablets and liquid, and also in combination with meprobamate or methyltestosterone.

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Squibb Announces Chemipen

Squibb Alpha-Pr[illegible]oxyethyl Pe[illegible]illin Potassium

new chemically improved penicillin which provides the highest blood levels that are obtainable with oral penicillin therapy



As a pioneer and leader in penicillin therapy for more than a decade, Squibb is pleased to make Chemipen, a new chemically improved oral penicillin, available for clinical use.

With Chemipen it becomes possible as well as convenient for the physician to achieve and maintain higher blood levels—with greater speed—than those produced with comparable therapeutic doses of potassium penicillin V. In fact, Chemipen is shown to have a 2:1 superiority in producing peak blood levels over potassium penicillin V.*

Extreme solubility may contribute to the higher blood levels that are so notable with Chemipen.* Equally notable is the remarkable resistance to acid decomposition (Chemipen is stable at 37°C. at pH 2 to pH 3), which in turn makes possible the convenience of oral treatment.

And the economy for your patients will be of particular interest—Chemipen costs no more than comparable penicillin V preparations.

Dosage: Doses of 125 mg. (200,000 u.) or 250 mg. (400,000 u.), t.i.d., depending on the severity of the infection. The usual precautions must be carefully observed with Chemipen, as with all penicillins. Detailed information is available on request from the Professional Service Department.

Supply: Chemipen Tablets of 125 mg. (200,000 u.) and 250 mg. (400,000 u.), bottles of 24 tablets. Chemipen Syrup (cherry-mint flavored, nonalcoholic), 125 mg. per 5 cc., 60 cc. bottles.

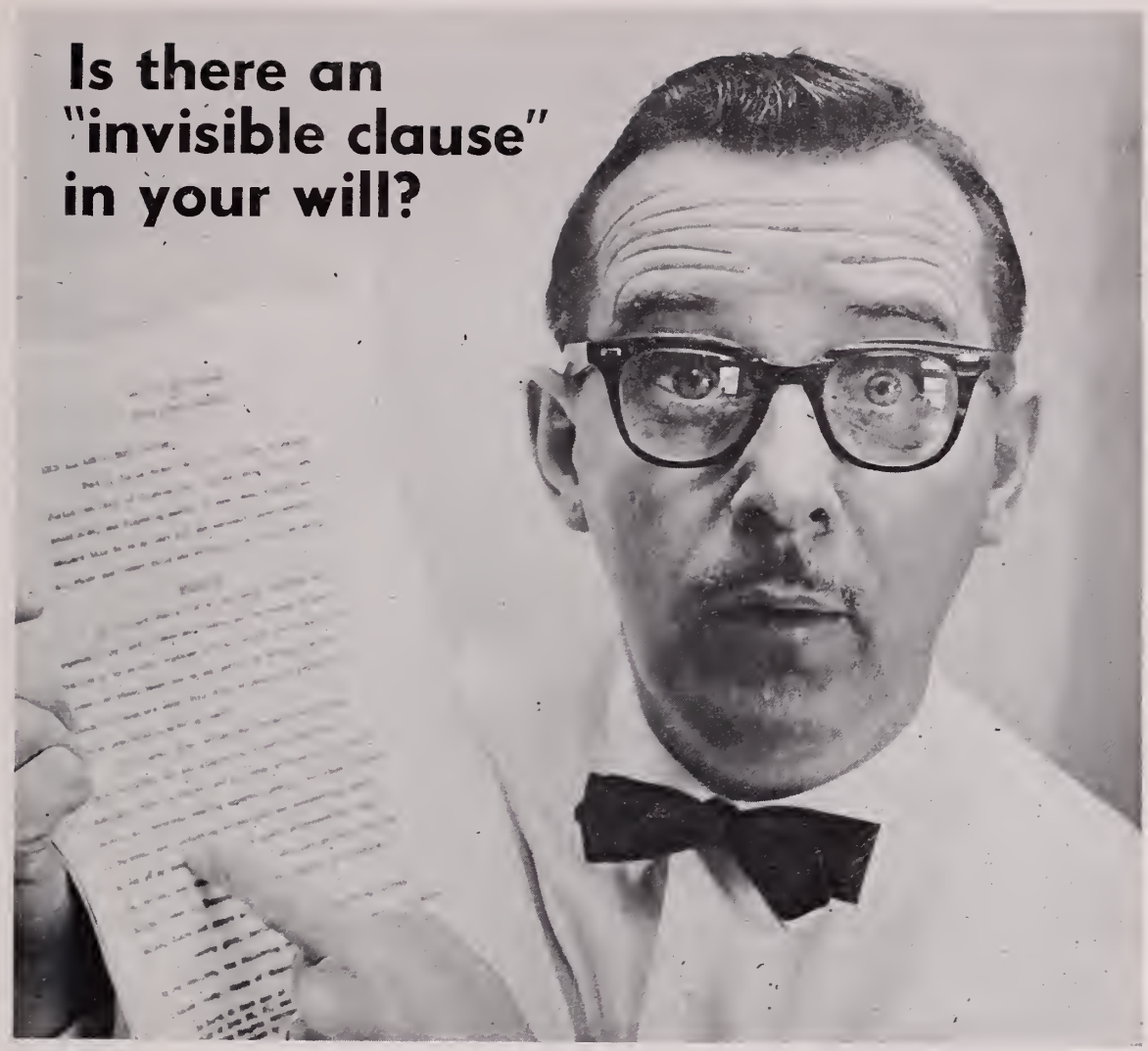
*Knudsen, E. T., and Rolinson, G. N.: Lancet 2:1105 (Dec. 19) 1959.

CHEMIPEN IS A SQUIBB TRADEMARK.

SQUIBB

Squibb Quality—the Priceless Ingredient

Is there an "invisible clause" in your will?



"I hereby accidentally bequeath $20,000 of my estate to the tax collector."

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You won't find those clauses written in any will. But they may be there anyway. You can guard against this problem with a trust under your will. Let us explain the family security and tax saving advantages of a trust under your will. A conference costs you nothing, yet it may save your estate many thousands of dollars.

Send for Liberty's free booklet "A Trust Under Your Will — for family security and tax saving." There's no obligation.

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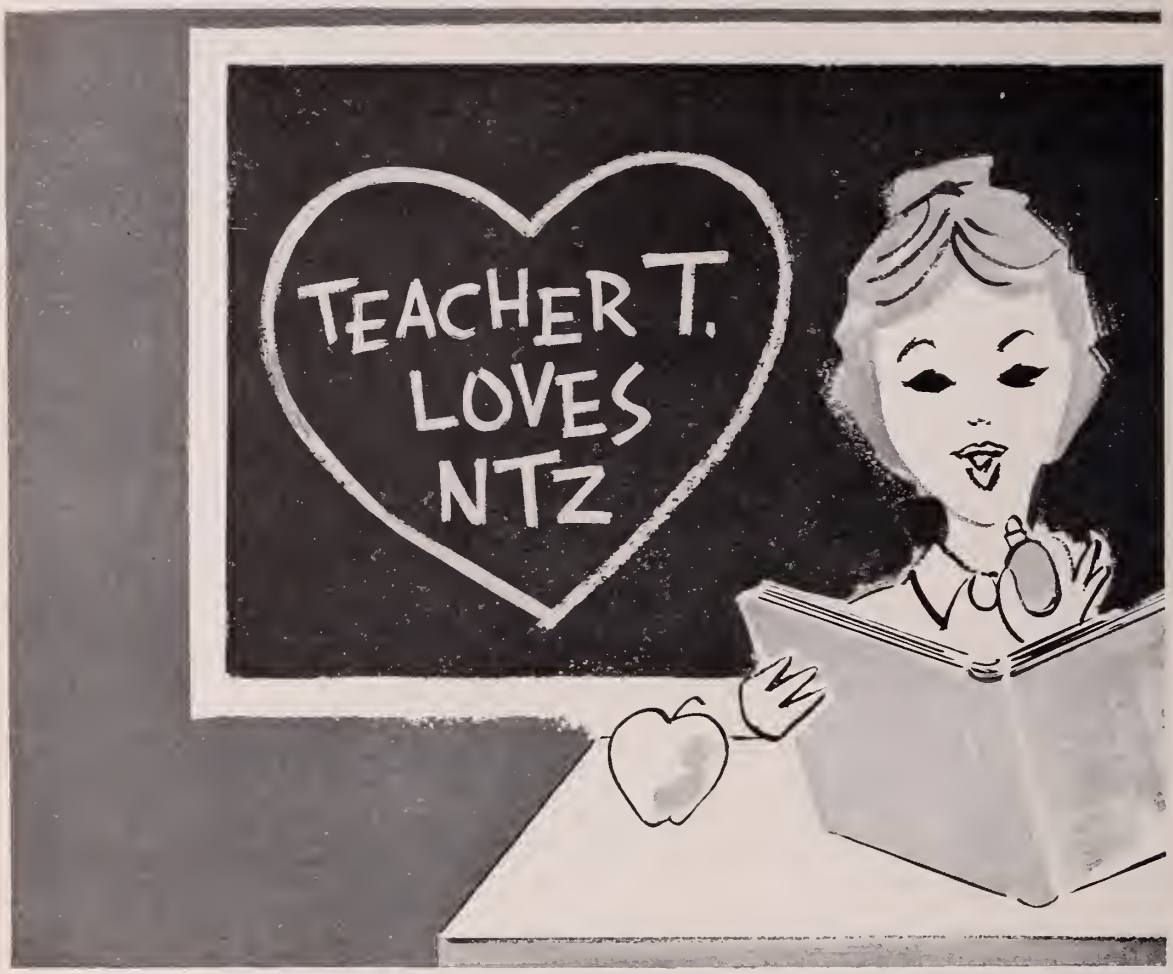
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the lasting relief from colds
puts NTZ Nasal Spray
in a class by itself.**

NTZ® NASAL SPRAY

*20 cc. spray bottles;
also 1 oz. bottles with dropper*

Neo-Synephrine® HCl, 0.5%
—unexcelled decongestant—

Thenfadil® HCl, 0.1%
—topical antihistaminic—

Zephiran® Cl, 1:5000
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NTZ, Neo-Synephrine (brond of phenylephrine), Thenfadil (brond of thenyldiomine) and Zephiron (brond of benzalkonium, os chloride, refined), trademarks reg. U. S. Pat. Off.

Potentiated Relief for Colds...Sinusitis...Hay Fever



When blood pressure must come down

When you see symptoms of hypertension such as dizziness, headache, and fainting your patient is a candidate for Serpasil-Apresoline. Even when single-drug therapy fails, Serpasil-Apresoline frequently can bring blood pressure down to near-normal levels, reduce rapid heart rate, allay anxiety.

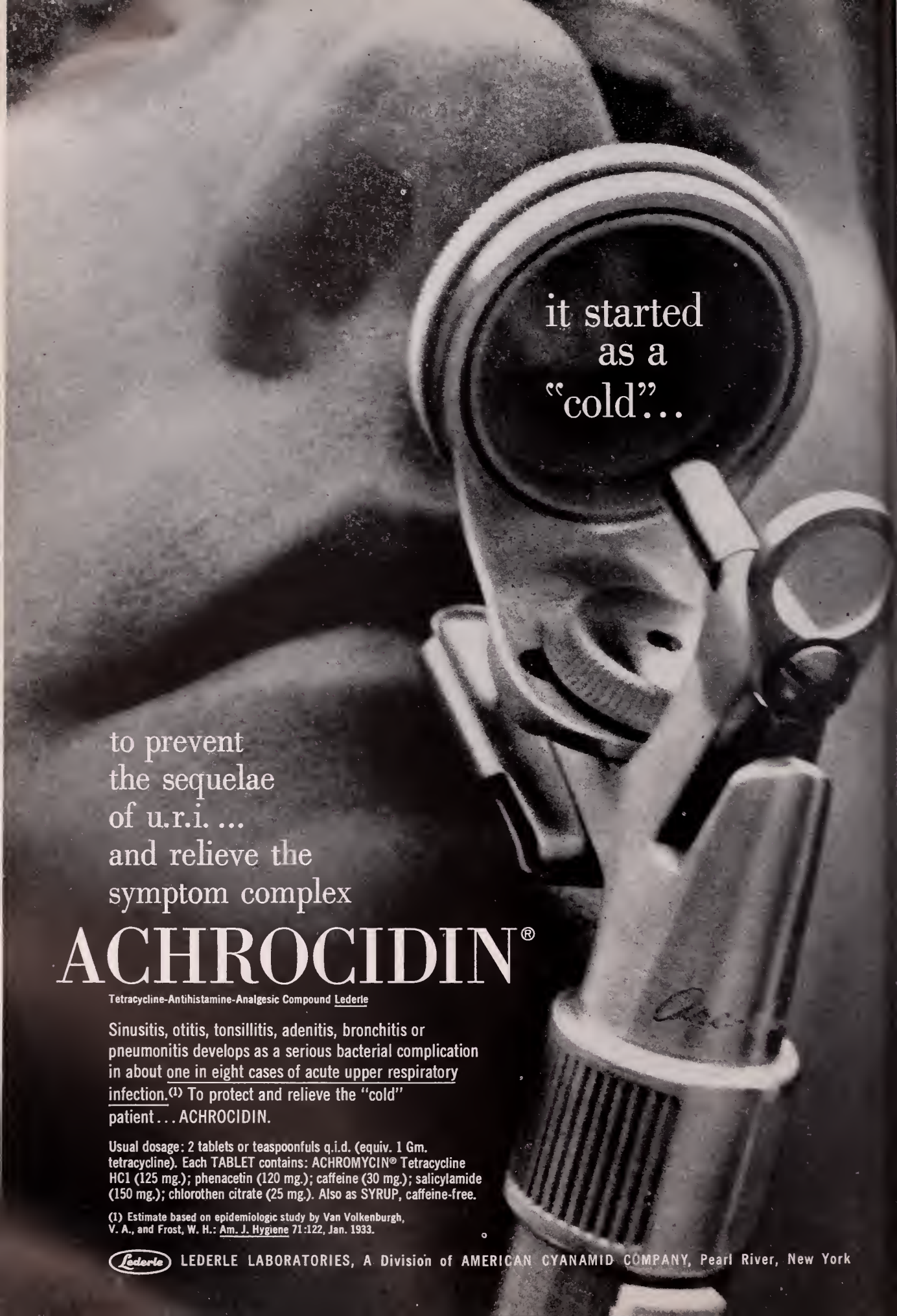
SUPPLIED: Tablets #2 (standard-strength, scored), each containing 0.2 mg. Serpasil and 50 mg. Apresoline hydrochloride; Tablets #1 (half-strength, scored), each containing 0.1 mg. Serpasil and 25 mg. Apresoline hydrochloride.

SERPASIL®-APRESOLINE®

hydrochloride (reserpine and hydralazine hydrochloride CIBA)

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CIBA
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to prevent
the sequelae
of u.r.i. ...
and relieve the
symptom complex

ACHROCIDIN®

Tetracycline-Antihistamine-Analgesic Compound Lederle

Sinusitis, otitis, tonsillitis, adenitis, bronchitis or pneumonitis develops as a serious bacterial complication in about one in eight cases of acute upper respiratory infection.(1) To protect and relieve the "cold" patient... ACHROCIDIN.

Usual dosage: 2 tablets or teaspoonfuls q.i.d. (equiv. 1 Gm. tetracycline). Each TABLET contains: ACHROMYCIN® Tetracycline HCl (125 mg.); phenacetin (120 mg.); caffeine (30 mg.); salicylamide (150 mg.); chlorothen citrate (25 mg.). Also as SYRUP, caffeine-free.

(1) Estimate based on epidemiologic study by Van Volkenburgh, V. A., and Frost, W. H.: Am. J. Hygiene 71:122, Jan. 1933.

Lederle LEDERLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Tetracycline Phosphate Complex (TETREX®)
U.S. PAT. NO. 2,791,609
in the Therapy of

ACUTE PHARYNGITIS, ESPECIALLY WITH LYMPHADENITIS

Ideally, selection of the proper antibiotic for treatment of acute pharyngitis should await the laboratory reports on the susceptibility of the infecting bacteria. But the busy practitioner who sees many patients a day during the upper respiratory infection season may sometimes find it difficult to avoid the empirical choice of an antibiotic. Unfortunately, this practice may sometimes result in therapeutic failure.

No matter what the pressure of the immediate situation, it is worthwhile to consider taking a bacterial specimen from the infected pharynx for culture and sensitivity studies before starting treatment. Thus, a rational basis will be provided for changing the antibiotic should the first choice prove ineffective.

Which Antibiotic?

All other things being equal, the drug of choice is the one to which the pathogen is most susceptible. But if the exigencies of the situation force the physician to a prompt use of antibiotic, a broad-spectrum preparation that produces immediate high blood levels (e.g., tetracycline phosphate complex, TETREX) probably has the best chance of controlling the pathogen.

Later, the laboratory report frequently may indicate that any one of several antibiotic agents would be equally effective against the particular microorganism in question. In such a case other factors such as frequency and severity of side effects, sensitizing potential and toxicity should be considered.

If the acute pharyngitis in question should be due to gram-negative Klebsiella[1], penicillin will be of no value, nor will erythromycin be effective. However, this organism is susceptible to tetracycline. If the pathogen should turn out to be gram-positive Streptococcus or Staphylococcus, then penicillin, erythromycin, and tetracycline may all be effective against it.

Penicillin, however, in addition to having a limited spectrum, also causes many minor and some serious sensitivity reactions. In a recent survey[2] it was found that penicillin produced severe skin reactions. But most important was the observation that anaphylactic shock, with a fatality rate of about 9 per cent, was the most frequent serious reaction. Such severe reactions are almost always associated with parenteral administration.

The tetracyclines (e.g., TETREX) have the advantages of a broad range of antimicrobial activity and low toxicity. And in addition, the physician does not have to trouble himself or his patients with repeated blood studies when he prescribes TETREX. Minor reactions such as gastric upsets or mild skin rashes occur occasionally. The most serious side effects are staphylococcal and monilial overgrowth, but these are rare and can be adequately controlled.

Some Microorganisms Susceptible[a] *to Tetracycline* (TETREX)[b]

Streptococcus; Staphylococcus; Pneumococcus; Gonococcus; Meningococcus; *C. diphtheriae; B. anthracis; E. coli;* Proteus; *A. aerogenes; K. pneumoniae;* Shigella; Brucella; *P. tularensis; H. influenzae; T. pallidum;* Rickettsiae; Viruses of psittacosis and ornithosis, lymphogranuloma inguinale, primary atypical pneumonia; *E. histolytica; D. granulomatosis.*

[a] Some strains are not susceptible.

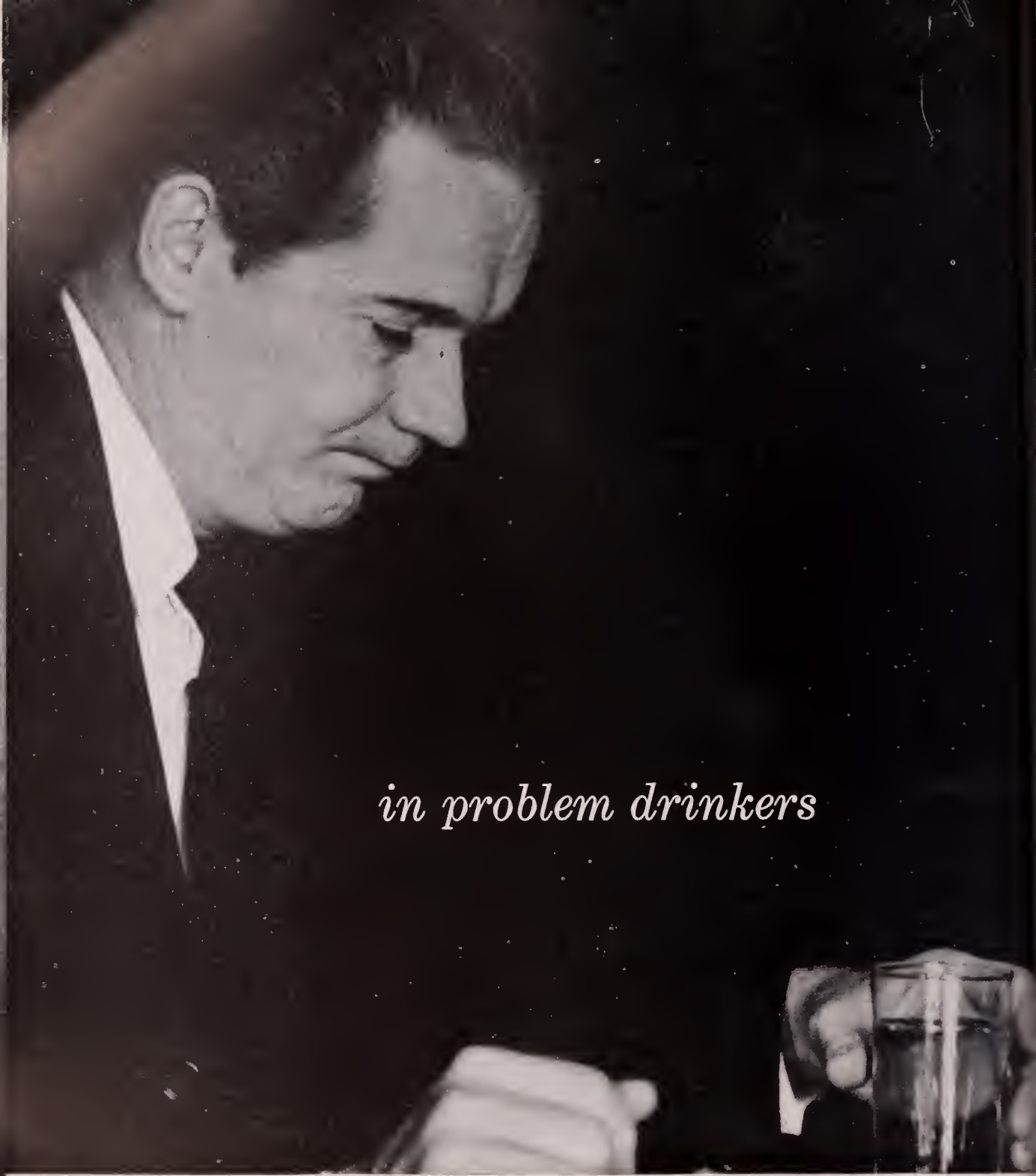
[b] Table adapted from Goodman, L. S., and Gilman, A.: The Pharmaceutical Basis of Therapeutics, 2nd edition, New York, The Macmillan Co., 1956, pp. 1322-1323.

High blood, body fluid, and tissue levels of active drug are quickly attained when the new phosphate preparation of tetracycline (TETREX) is used.

The semisynthetic tetracyclines have been in constant use since they were introduced in 1952. They have been proved clinically and have established themselves as safe, effective, and valuable antibiotic agents. But the final decision, the choice of agent, and the control of therapy must remain where it has always been, in the hands of the individual physician.

References: 1. Zinsser, H.: A Textbook of Bacteriology. 11th edition, New York, Appleton-Century-Crofts, 1957, p. 409. 2. Welch, H.; Lewis, C. H.; Weinstein, H. I., and Boeckman, B. B.: Severe reactions to antibiotics. A nationwide survey. Antibiotic Med. & Clin. Ther. 4:800 (December) 1957.

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Vistaril®

hydroxyzine pamoate

dispels tension...
maintains tranquility

When tension and anxiety "drive him to drink," the problem drinker often finds that VISTARIL, by maintaining tranquility, restores perspective and helps him accept counsel more readily.

VISTARIL has demonstrated a wide margin of safety even in large doses (300-400 mg. daily) over prolonged periods. Clinical studies of alcoholism have shown that VISTARIL produces no significant depression of blood pressure, pulse rate, or respiration in chronic drinkers.

Capsules—25, 50, and 100 mg. *Parenteral Solution* (as the HCl)—25 mg. per cc., 10 cc. vials and 2 cc. Steraject® Cartridges; 50 mg. per cc., 2 cc. ampules.

Professional literature available on request from the Medical Department, Pfizer Laboratories, Div., Chas. Pfizer & Co., Inc., Brooklyn 6, New York

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in arthritis and allied disorders

proved by a decade of experience

Ten years of experience in countless cases—more than 1700 published reports—have now established the eminence of Butazolidin among the potent non-hormonal antiarthritic agents.

Repeatedly it has been demonstrated that Butazolidin:
Within 24 to 72 hours produces striking relief of pain.
Within 5 to 10 days affords a marked improvement in mobility and a significant subsidence of inflammation with reduction of swelling and absorption of effusion.

Even when administered over months or years Butazolidin does not provoke tolerance nor produce signs of hormonal imbalance.

Butazolidin® brand of phenylbutazone: Red-coated tablets of 100 mg. Butazolidin® Alka: Capsules containing Butazolidin® 100 mg.; dried aluminum hydroxide gel 100 mg.; magnesium trisilicate 150 mg.; homatropine methylbromide 1.25 mg.

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Geigy

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BAD DIGESTION INCLINES ONE TO SKEPTICISM, INCREDULITY, BREEDS BLACK FANCIES AND THOUGHTS OF DEATH JOSEPH CONRAD

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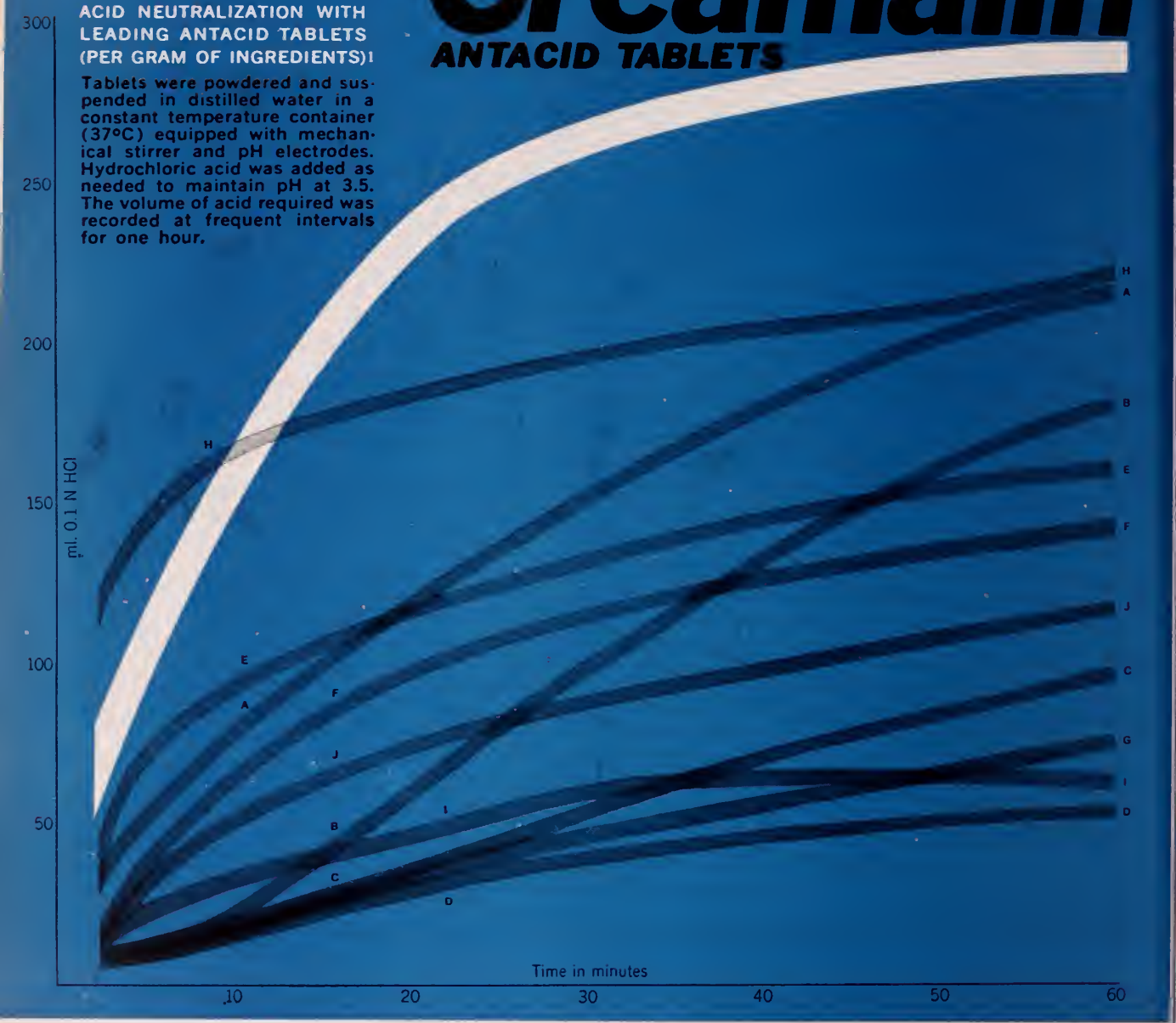
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More gastric acid neutralized faster...with new Creamalin®

ANTACID TABLETS

ACID NEUTRALIZATION WITH LEADING ANTACID TABLETS (PER GRAM OF INGREDIENTS)[1]

Tablets were powdered and suspended in distilled water in a constant temperature container (37°C) equipped with mechanical stirrer and pH electrodes. Hydrochloric acid was added as needed to maintain pH at 3.5. The volume of acid required was recorded at frequent intervals for one hour.



GREATLY HEIGHTENED REACTIVITY to acid characterizes the action of New Creamalin Antacid Tablets.[1,2] They act faster and longer than other leading tablets and neutralize considerably more acid.[1] These tablets provide virtually the same effects as a liquid[2] with the convenience of a tablet. New Creamalin tablets give faster, greater and more prolonged relief.

NOT CONSTIPATING, New Creamalin Antacid Tablets will not produce "acid rebound" or alkalosis. They have a pleasant taste.

EACH NEW CREAMALIN ANTACID TABLET contains 320 mg. of specially processed, highly reactive, short polymer dried aluminum hydroxide gel (stabilized with hexitol), with 75 mg. of magnesium hydroxide.

Adult dosage: Gastric hyperacidity—2 to 4 tablets as necessary. Peptic ulcer or gastritis—2 to 4 tablets every two to four hours. Tablets may be chewed, swallowed whole with water or milk, or allowed to dissolve in the mouth.

How Supplied: Bottles of 50, 100, 200 and 1000.

1. Hinkel, E. T., Jr.; Fisher, M. P., and Tainter, M. L.: *J. Am. Pharm. A. (Scient. Ed.)* 48:380, July, 1959. 2. Hinkel, E. T., Jr.; Fisher, M. P., and Tainter, M. L.: *J. Am. Pharm. A. (Scient. Ed.)* 48:384, July, 1959.

Winthrop LABORATORIES New York 18, N. Y.

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FOR PEPTIC ULCER · GASTRITIS · HYPERACIDITY



whenever there is inflammation, swelling, pain

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STREPTOKINASE-STREPTODORNASE LEDERLE

BUCCAL *Tablets*

conditions for a fast comeback...

as in acute hemorrhoids...

SUNDAY, 9 A.M.: VARIDASE for painful thrombotic hemorrhoid. 2:30 P.M.: pain greatly reduced, less swelling and inflammation.

MONDAY: size down to small tab; acute inflammation disappeared.*

VARIDASE activates natural fibrinolytic factors, to limit undesirable inflammatory response and speed healing.

Dramatic reduction of pain is often the first sign of improvement; swelling and redness rapidly diminish. Drugs and natural regenerative factors readily penetrate the inflammatory barrier to effect total remission faster... in trauma or infection.

VARIDASE Buccal Tablets contain:
10,000 Units Streptokinase, 2,500 Units Streptodornase.
Supplied: Boxes of 24 and 100 tablets

*Peterman, R. A.: Clinical report cited with permission.

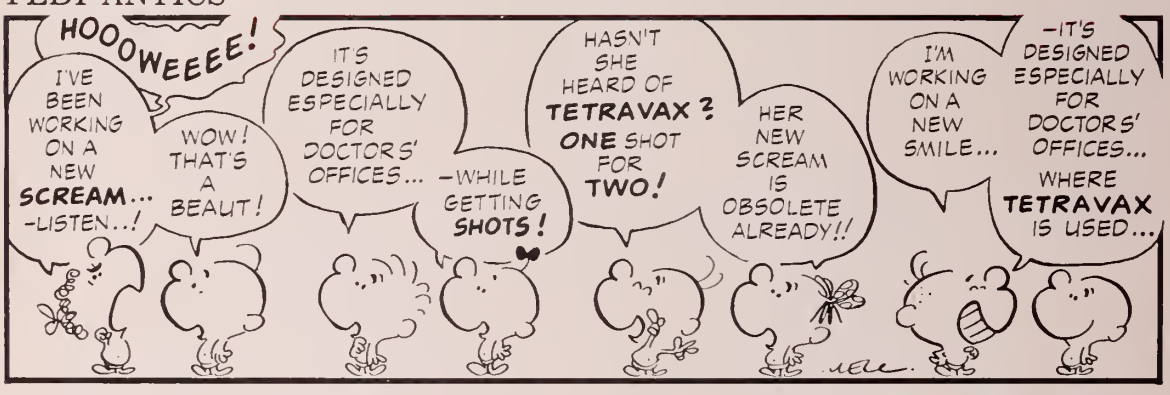
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FOR SIMULTANEOUS IMMUNIZATION AGAINST 4 DISEASES:

Poliomyelitis-Diphtheria-Pertussis-Tetanus

PEDI-ANTICS



TETRAVAX®

DIPHTHERIA AND TETANUS TOXOIDS WITH PERTUSSIS AND POLIOMYELITIS VACCINES

now you can immunize against more *diseases...with* fewer *injections*

Dose: 1 cc.
Supplied: 9 cc. vials in *clear plastic* cartons. Package circular and material in vial can be examined without damaging carton. Expiration date is on vial for checking even if carton is discarded.



For additional information, write Professional Services, Merck Sharp & Dohme, West Point, Pa.

TETRAVAX IS A TRADEMARK OF MERCK & CO., INC.

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in every arthritic state
long-term therapy
with maximum relief of pain
without steroids

Buffered

Pabirin®

Tablets

improved salicylate therapy

SMITH-DORSEY • *a division of The Wander Company* • *Lincoln, Nebraska*

in the **"COMMON COLD"**
when self-medication has delayed medical attention...

...and has risked upper respiratory complications



COSA-TETRACYDIN® **CAPSULES**

Cosa-Tetracyn® — analgesic — antihistamine compound

act quickly to

- *control secondary infection*
- *alleviate cold symptoms*

each capsule contains:

Cosa-Tetracyn	125 mg.
phenacetin	120 mg.
caffeine	30 mg.
salicylamide	150 mg.
buclizine HCl	15 mg.

average adult dose: 2 capsules q. i. d.

Pfizer *Science for the world's well-being*

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Each of the babies pictured on this page was borne by a mother with a *documented* previous history of true habitual abortion, who was treated with DELALUTIN during the pregnancy leading to this birth

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DELALUTIN

SQUIBB HYDROXYPROGESTERONE CAPROATE

Improved Progestational Therapy



Garden City, N. Y.



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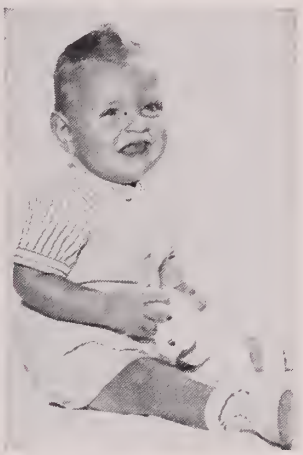
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DELALUTIN offers these advantages over other progestational agents

• long-acting sustained therapy • more effective in producing and maintaining a completely matured secretory endometrium • no androgenic effect • more concentrated solution requiring injection of less vehicle • unusually well-tolerated, even in large doses • fewer injections required • low viscosity makes administration easy

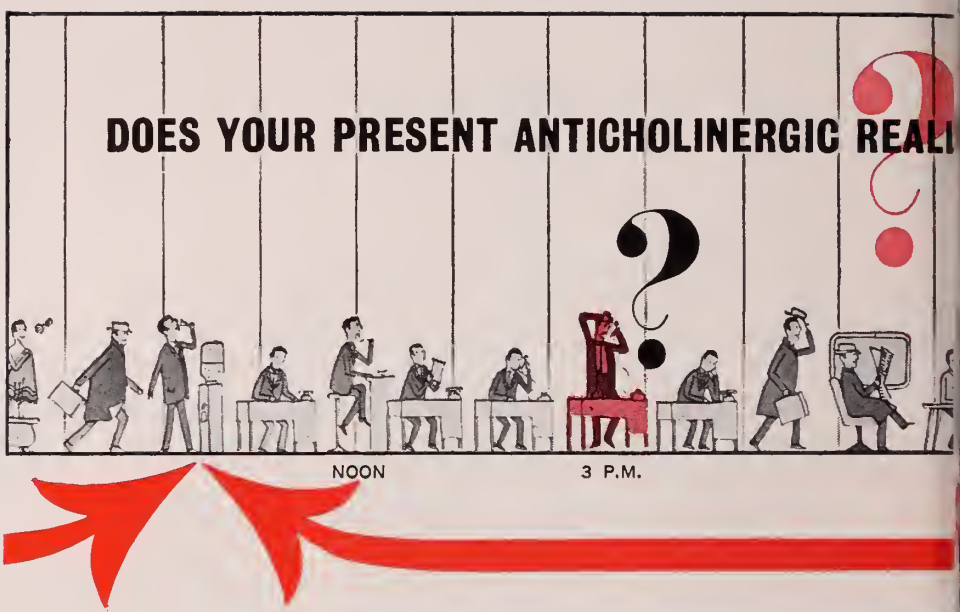
Complete information on administration and dosage is supplied in the package insert

Supply: Vials of 2 and 10 cc., each containing 125 mg. of hydroxyprogesterone caproate in benzyl benzoate and sesame oil.

SQUIBB *Squibb Quality—The Priceless Ingredient*

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DOES YOUR PRESENT ANTICHOLINERGIC REALI



The test—you might say the acid test—of an anticholinergic is simple: will it protect your patient from hyperacidity around the clock, **even while he sleeps.** The weakness of t.i.d. or q.i.d. preparations is well recognized; but even some "b.i.d." encapsulations may be unreliable. McHardy, for instance, found a "widely variable duration of action, definitely less than that anticipated" in the "sustained," "delayed," and "gradual release" anticholinergics he studied.[1]

COMPARE THE DATA ON ENARAX . . . the new combination of an **inherently** long-acting anticholinergic (oxyphencyclimine) and Atarax, the non-secretory tranquilizer. Note the effectiveness of oxyphencyclimine:

OBSERVE THE OXYPHENCYCLIMINE REPORTS . . .

McHardy: "[Oxyphencyclimine] has proved to be an excellent sustained-action anticholinergic in our study of this agent over a period of eighteen months."[1]

Kemp: ". . . for the majority of patients, one tablet every 12 hours provided adequate control. This characteristic long action . . . may constitute an advantage of this drug as compared to coated 'long-acting' preparations of other compounds."[3]

Add Atarax to this 12-hour anticholinergic. The resulting combination—ENARAX—now gives relief from emotional stress, in addition to a reduction of spasm and acid. Atarax does not stimulate gastric secretion. No serious adverse clinical reaction has ever been documented with Atarax.

LOOK AT THE RESULTS WITH ENARAX[4,5]:

Does the medication you now prescribe assure you of all these benefits? If not, why not put your next patient with peptic ulcer or G.I. dysfunction on therapy that **does.**

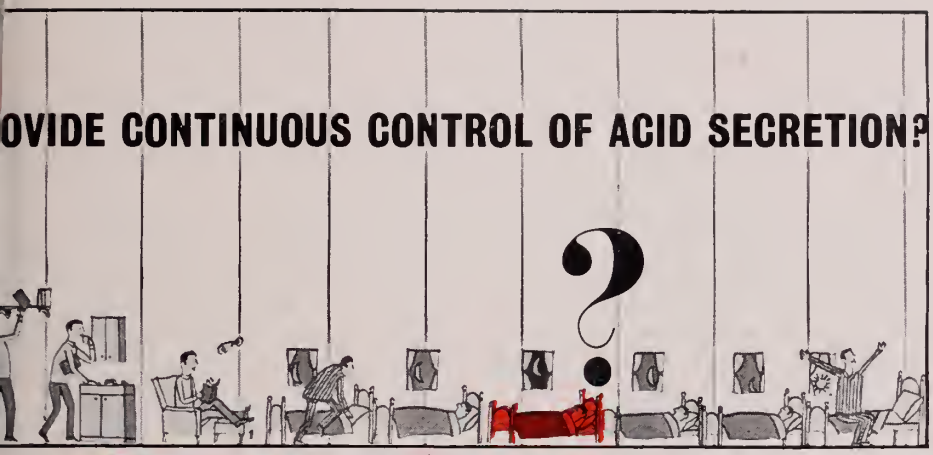
ENARAX®



(oxyphencyclimine plus ATARAX®)

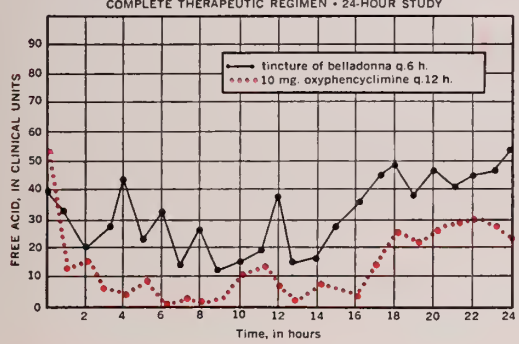
A SENTRY FOR THE G.I. TRACT

OVIDE CONTINUOUS CONTROL OF ACID SECRETION?



"Prolonged periods of achlorhydria" after 10 mg. oxyphencyclimine q. 12 h.[2]

MEAN GRAPH OF GASTRIC ACIDITY IN 4 PATIENTS RECEIVING COMPLETE THERAPEUTIC REGIMEN • 24-HOUR STUDY



Clinical Diagnosis: Peptic Ulcer—Gastritis—Gastroenteritis—Colitis—Functional Bowel Syndrome—Duodenitis—Hiatus Hernia (symptomatic)—Irritable Bowel Syndrome—Pylorospasm—Cardiospasm—Biliary Tract Dysfunctions—and Dysmenorrhea.

Clinical Results: Effective in over 92% of cases.

As for Safety: "Side reactions were uncommon, usually no more than dryness of the mouth...."[4]

Each ENARAX tablet contains:

Oxyphencyclimine HCl ..... 10 mg.
Hydroxyzine (ATARAX®) ..... 25 mg.

Dosage: One-half to one tablet twice daily—preferably in the morning and before retiring. The maintenance dose should be adjusted according to therapeutic response. Use with caution in patients with prostatic hypertrophy and with ophthalmological supervision only in glaucoma.

Supplied: In bottles of 60 black-and-white scored tablets.

References: 1. McHardy, G., et al.: J. Louisiana M. Soc. 111:290 (Aug.) 1959. 2. Steigmann, F.: Study conducted at Cook County Hospital, Chicago, Illinois, in press. 3. Kemp, J. A.: Antibiotic Med. & Clin. Therapy 6:534 (Sept.) 1959. 4. Leming, B. H., Jr.: Clin. Med. 6:423 (Mar.) 1959. 5. Data in Roerig Medical Department files.

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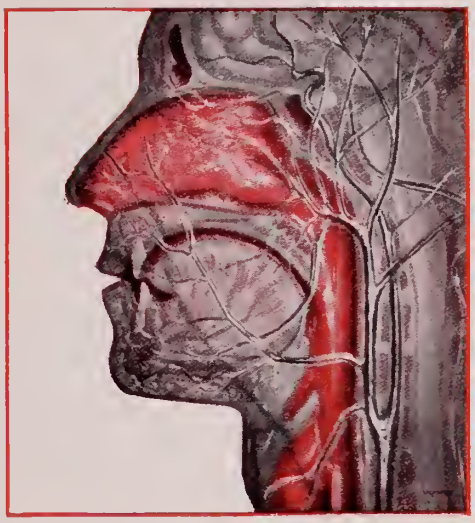
1. Lippmann, O.: Arch. Ophth. **57**:339, March 1957.
2. Gordon, D.M.: Am. J. Ophth. **46**:740, November 1958.

supplied: 0.5% Sterile Ophthalmic Solution NEO-HYDELTRASOL (with neomycin sulfate) and 0.5% Sterile Ophthalmic Solution HYDELTRASOL*. In 5 cc. and 2.5 cc dropper vials. Also available as 0.25% Ophthalmic Ointment NEO-HYDELTRASOL (with neomycin sulfate) and 0.25% Ophthalmic Ointment HYDELTRASOL. In 3.5 Gm. tubes.

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MSD MERCK SHARP & DOHME Division of Merck & Co., Inc., Philadelphia 1, Pa.

reaches all nasal and paranasal membranes *systemically*[1]



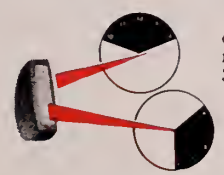
Pharmacologically balanced formula for prompt symptomatic relief

- in nasal and paranasal congestion
- in sinusitis and postnasal drip
- in allergic reactions of the upper respiratory tract

Triaminic*[2,3] *is safer and more effective than topical medication

- transported systemically to *all* respiratory membranes
- provides longer-lasting relief
- presents no problem of rebound congestion
- avoids "nose drop addiction"

Relief is prompt and prolonged because of this special timed-release action:



first— the outer layer dissolves within minutes to produce 3 to 4 hours of relief

then— the core disintegrates to give 3 to 4 *more* hours of relief

Each Triaminic timed-release Tablet provides:
Phenylpropanolamine HCl.....50 mg.
Pheniramine maleate.....25 mg.
Pyrilamine maleate.....25 mg.

Dosage: 1 tablet in the morning, midafternoon and at bedtime. In postnasal drip, 1 tablet at bedtime is usually sufficient.

Each timed-release Triaminic Juvelet® provides: ½ the formulation of the Triaminic Tablet.

Dosage: 1 Juvelet in the morning, midafternoon and at bedtime.

Each tsp. (5 ml.) of Triaminic Syrup provides: ¼ the formulation of the Triaminic Tablet.

Dosage (to be administered every 3 or 4 hours): *Adults* — 1 or 2 tsp.; *Children 6 to 12* — 1 tsp.; *Children 1 to 6* — ½ tsp.; *Children under 1* — ¼ tsp.

1. Fabricant, N. D.: E.E.N.T. Monthly *37*:460 (July) 1958.
2. Lhotka, F. M.: Illinois M. J.: *112*:259 (Dec.) 1957.
3. Farmer, D. F.: Clin. Med. *5*:1183 (Sept.) 1958.

the leading oral nasal decongestant...

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timed-release tablets and juvelets
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The first synthetic penicillin available for general clinical use

FOR YOUR NEXT PATIENT WHERE PENICILLIN IS INDICATE

SYNC

PEAK BLOOD LEVELS TWICE AS HIGH AS WITH POTASSIUM PENICILLIN V

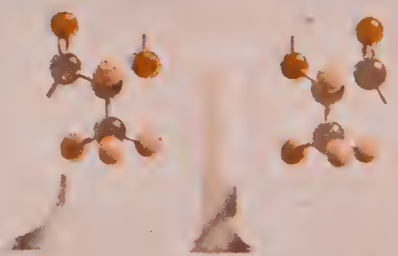
ORAL ROUTE PROVIDES HIGHER PEAK BLOOD LEVELS THAN INTRAMUSCULAR PENICILLIN G

IMPROVED ANTIBIOTIC ACTION FROM ISOMERIC COMPLEMENTARITY





ONSIDER THESE 6 IMPORTANT THERAPEUTIC ATTRIBUTES OF



TM

POTASSIUM PENICILLIN-152

ANTIBIOTIC ACTIVITY DIRECTLY PROPORTIONAL TO ORAL DOSE	*REDUCED RATE OF INACTIVATION BY STAPH PENICILLINASE*	*MANY STAPH STRAINS MORE SENSITIVE TO SYNCILLIN IN VITRO*

BRISTOL





FOR HIGHLY EFFECTIVE THERAPY OF THE LARGE VARIETY OF INFECTIONS CAUSED BY SUSCEPTIBLE PATHOGENS...NEW

SYNC

Significance of complementary action of isomers in SYNCILLIN

The antibiotic effect of the clinically available mixture, SYNCILLIN, is greater than that of either of its two component isomers alone against many important pathogens, including some penicillin-resistant staphylococci. This phenomenon has been described as *Isomeric Complementarity*.

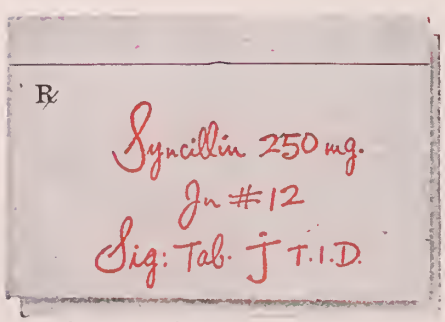
Significance of higher blood levels with SYNCILLIN

Higher blood levels may be of value with organisms of only moderate penicillin sensitivity where doubling the blood concentration may be essential for effective bactericidal action. In addition, these higher levels may be necessary where there is infection in areas with a poor blood supply. Under these circumstances a higher blood concentration may provide the increased diffusion pressure required to deliver adequate amounts to the tissue. Also, antibiotic activity of SYNCILLIN is directly proportional to oral dosage. Increasing the dosage may, therefore, enhance the drug's effectiveness in certain cases.

Efficacy of SYNCILLIN against staphylococci and other resistant organisms

Studies have shown that SYNCILLIN is effective *in vitro* against 60 to 75% of hospital "staph" strains, while penicillin G and penicillin V are now effective against only 30 to 50%.[1, 2] Therefore, if clinical judgment indicates the use of penicillin, SYNCILLIN would be expected to be the most effective. However, since some strains are still resistant to SYNCILLIN as well as to the other penicillins, cultures and sensitivity tests should be performed where indicated by clinical judgment.

There have recently been reports of decreased efficacy of penicillin in streptococcal[3] and gonococcal[4, 5] infections. The emergence of penicillin-resistant gonococci appears to be associated with an increase in the incidence of gonorrhea all over the world. When a less sensitive strain is encountered the higher blood levels produced by SYNCILLIN may be most helpful.



major therapeutic advantages accompany molecular asymmetry

I L L I N™

Relation of intermittent high blood levels of SYNCILLIN to antibacterial efficacy

SYNCILLIN, like all clinically available penicillins, is bactericidal. Periodic high blood concentrations may be sufficient to permit complete eradication of sensitive pathogens. According to Eagle,[6] "Soon after penicillin attains effective concentrations, the bacteria cease multiplying; and the bacteriostatic effect persists for a number of hours after penicillin has fallen to concentrations that are wholly ineffective. . . . The therapeutic significance of this postpenicillin recovery period is enhanced by the fact that the recovering bacteria, damaged but not killed by the previous exposure to penicillin, are abnormally susceptible to the host defenses. In consequence, the bactericidal process *in vivo* continues for many hours after the drug itself has fallen to ineffective concentrations."

Reduced rate of inactivation of SYNCILLIN by staph penicillinase

Bacterial resistance to penicillin has been attributed to the action of penicillin-inactivating enzymes produced by the invading organisms. SYNCILLIN is less affected by staphylococcal penicillinase than either of its component isomers. Further, SYNCILLIN is shown to be less inactivated by this enzyme than penicillin V or penicillin G. Penicillinase from *B. cereus* likewise inactivates SYNCILLIN less rapidly than penicillin V or G.

Indications: SYNCILLIN is recommended in the treatment of infections caused by pneumococci, streptococci, gonococci, corynebacteria, and penicillin-sensitive staphylococci. In addition, SYNCILLIN is effective against certain strains of staphylococci resistant to other penicillins. SYNCILLIN, like other oral penicillins, is not recommended at the present time in deep-seated or chronic infections, subacute bacterial endocarditis, meningitis, or syphilis.

Dosage: 125 mg. or 250 mg. three times daily, depending on the severity of infection. Larger doses (e.g., 500 mg. t.i.d.) may be used for more severe infections. SYNCILLIN may be administered without regard to meals. Beta hemolytic streptococcal infections should be treated with SYNCILLIN for at least ten days.

Precautions: At the present time it is not possible to draw definite conclusions regarding the incidence of allergenicity to SYNCILLIN or its cross-allergenicity with natural penicillins. *Therefore, the usual precautions for oral penicillin therapy should always be observed.* Patients with histories of asthma, hay fever, urticaria, or previous reactions to penicillin should be watched with special care. Administration of oral penicillin, in rare instances, may provoke acute anaphylaxis, particularly in penicillin-sensitive individuals.

Diarrhea has been reported occasionally following heavy dosage. If this occurs, lengthen the interval between dosages.

If superinfection occurs during therapy, appropriate measures should be taken. Since some strains of staphylococci are resistant to SYNCILLIN as well as to other penicillins, cultures and sensitivity tests should be performed where indicated by clinical judgment. As is true with all antibiotics, clinical response does not always correlate with laboratory bacterial sensitivity reports.

Supply: 125 and 250 mg. tablets, bottles of 25 and 100. 125 mg. powder for oral solution, 60 ml. vials.

References: 1. Wright, W. W.: Microbiology Report to Bristol Laboratories Inc. 2. Morigi, E. M. E.; Wheatley, W. B., and Albright, H.: Paper presented at the Seventh Antibiotic Symposium, November 4-6, 1959, Washington, D.C. 3. Editorial: New England J. Med. **261**:305 (Aug. 6) 1959. 4. King, A.: Lancet **1**:651 (March 29) 1958. 5. Epstein, E.: J.A.M.A. **169**:1055 (March 7) 1959. 6. Eagle, H. and Musselman, A. D.: J. Bact. **58**:475, 1949.

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the mood brightener

eases mental adjustment to menopause

NIAMID brightens the outlook of depressed menopausal patients — gradually helps them become alert, cheerful, relaxed, and better able to cope with their surroundings.

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NIAMID (brand of nialamide) is available as 25 mg. (pink) and 100 mg. (orange) scored tablets.

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DURATION



IN *ORAL* CONTROL OF PAIN

ACTS FASTER—usually within 5-15 minutes. **LASTS LONGER**—usually 6 hours or more. **MORE THOROUGH RELIEF**—permits uninterrupted sleep through the night. **RARELY CONSTIPATES**—excellent for chronic or bedridden patients.

AVERAGE ADULT DOSE: 1 tablet every 6 hours. May be habit-forming. Federal law permits oral prescription.

Each PERCODAN* Tablet contains 4.50 mg. dihydrohydroxycodeinone hydrochloride, 0.38 mg. dihydrohydroxycodeinone terephthalate, 0.38 mg. homatropine terephthalate, 224 mg. acetylsalicylic acid, 160 mg. phenacetin, and 32 mg. caffeine.

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Just one prescription for **Engran** Term-Pak
SQUIBB VITAMIN-MINERAL SUPPLEMENT *(270 tablets)*
calling for just one tablet per day will carry her through term to the six-week postpartum check-up. Thus, you help to assure a nutritionally perfect pregnancy, while providing the convenience and economy of the re-usable Term-Pak. Engran is also available in bottles of 100 tablets.

SQUIBB *Squibb Quality—The Priceless Ingredient*

'ENGRAN'® AND 'TERM-PAK'® ARE SQUIBB TRADEMARKS

SOMA RELIEVES PAIN in a unique way by modifying central perception of pain without abolishing natural defense reflexes.

SOMA RELAXES MUSCLE SPASM . . . approximately 8 times more potent than meprobamate or mephenesin.

PHYSICIANS' REPORTS:

"Marked pain-relieving effects of the new drug [SOMA] were seen in conditions involving muscle spasm and stiffness, whether acute or chronic. Relief from pain was usually rapid and sometimes dramatic." (90 patients.)
Kuge, T.: Submitted for publication.

"In 86 percent of the patients there were excellent or good results. . . . Relief of pain was noted by the patients' statements, by the diminished need for analgesic drugs, and by improved sleep." (154 patients.)
Wein, A. B.: The Use of Carisoprodol in Orthopedic Surgery and Rehabilitation. Proceedings of the Symposium on The Pharmacology and Clinical Usefulness of Carisoprodol. Wayne State University Press, Detroit, 1959, p. 156.

In a double-blind study, SOMA was reported to be "clinically effective to a highly significant degree." (92 patients.)
Cooper, C. D., and Epstein, J. H.: The Clinical Evaluation of Carisoprodol by a double-blind technique. Ibid. p. 97.

Notable safety—extremely low toxicity; no known contraindications; side effects are rare; drowsiness may occur, usually at higher dosage

Rapid action—starts to act quickly

Sustained effect—relief lasts up to 6 hours

Easy to use—usual adult dose is one 350 mg. tablet 3 times daily and at bedtime

Supplied—as white, coated, 350 mg. tablets, bottles of 50.
Also available for pediatric use: 250 mg. orange capsules, bottles of 50.

SOMA™

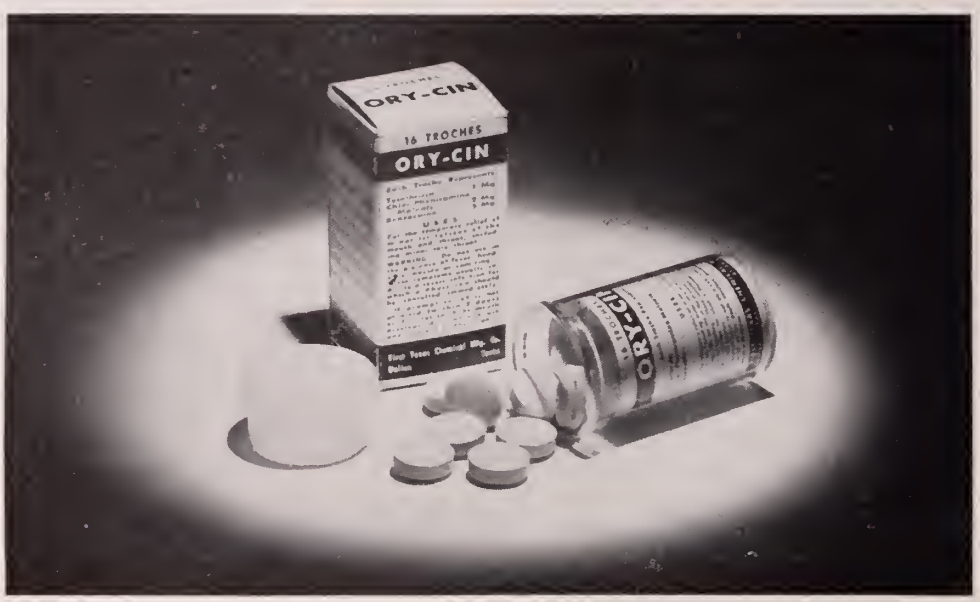
(carisoprodol Wallace)

BIBLIOGRAPHY: 1. Berger, F.M., Kletzkin, M., Ludwig, B.J., Margolin, S. and Powell, L. S.: J. Pharm. Exp. Ther. *127*:66 (Sept.) 1959. 2. Leake, Chauncey D.: Proceedings of the Symposium on The Pharmacology and Clinical Usefulness of Carisoprodol, Wayne State University Press, Detroit, 1959, p. 8. 3. Kestler, Otto: Ibid. p. 143. 4. Proctor, Richard C.: Ibid. p. 122. 5. Berger, Frank M., Ibid. p. 25. 6. Goodgold, Joseph, Hohmann, Thomas and Tajima, Toshihiro: Ibid. p. 66. 7. Gammon, George D. and Tucker, Samuel: Ibid. p. 70. 8. Baird, Henry W. and Menta, Dominic A.: Ibid. p. 85. 9. Cooper, C. David and Epstein, Jerome H.: Ibid. p. 97. 10. Korst, Donald R., Gerard, R. W., Miller, James G., Small, Iver F., Graham, I. J. and Winkelman, Eugene I : Ibid. p. 104. 11. Friedman, Arnold P.: Ibid. p. 115. 12. Trimpi, Howard D.: Ibid. p. 150. 13. Wein, Arthur B.: Ibid. p. 156. 14. Olds, James and Travis, R. P.: Ibid. p. 39. 15. Hess, Eckhard H., Polt, James M. and Goodwin, Elizabeth: Ibid. p. 51. 16. Phelps, Winthrop M.: Ibid. p. 131. 17. Spears, Catherine E.: Ibid. p. 138. 18. Hyde, L. P. and Hough, Charles E.: Ibid. p. 166. 19. Spears, Catherine E. and Phelps, Winthrop M.: Arch Pediat., *76*:287 (July) 1959. 20. Phelps, Winthrop M.: Arch. Pediat., *76*:243 (June) 1959. 21. Friedman, Arnold P.: Paper presented at Scientific Meeting, New York State Society of Industrial Medicine, Inc., New York, Sept. 30, 1959. 22. Frankel, Kalman: Ibid. 23. Fransway, Robert L.: Ibid. 24. Kuge, T.: Unpublished reports.

Literature and samples on request WALLACE LABORATORIES, New Brunswick, New Jersey

ORY-CIN

Antibiotic and Antihistaminie Medication for Common Mouth and Throat Infections



Infections of the mouth and throat are of common occurrence in everyday medical practice. In mild cases ORY-CIN troches will provide prompt and effective relief without internal medication. They may be used as a therapeutic adjuvant to internally administered antibiotics or sulfonamides in more severe conditions.

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ORY-CIN troches are designed to combat the infection, relieve the congestion, and soothe the local irritation. For this purpose three valuable ingredients are combined as follows:

Tyrothricin . . . . . **1 mg.**
Chlorpheniramine Maleate . . . . . **2 mg.**
Benzocaine . . . . . **5 mg.**

in a pleasantly flavored and readily soluble troche base.

DOSAGE AND ADMINISTRATION

One ORY-CIN troche should be held in the mouth and allowed to dissolve slowly. May be repeated every four hours.

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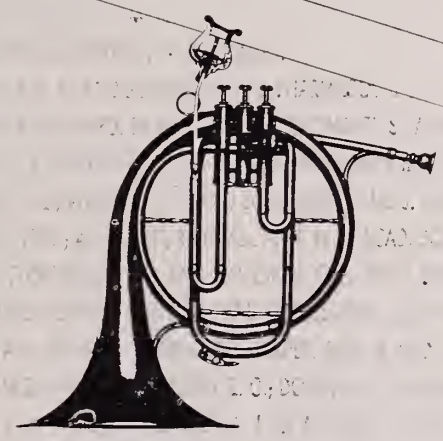
ORY-CIN troches are supplied in vials of 16 and bottles of 500.

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Only time and clinical acceptance truly define whether a drug is safe and effective. Of the many Rauwolfia compounds, there is one alkaloidal fraction capable of producing antihypertensive benefits with minimal side effects, purified alseroxylon complex (RAUTENSIN®).[1-6] This compound is less likely to cause such side effects as mental depression, lethargy, listlessness, and drowsiness consistently reported with reserpine.

IN MILD HYPERTENSION

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(Tablets containing 2 mg. purified alseroxylon complex)

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Rautensin bibliography: 1. WRIGHT, W. T., JR.; POKORY, C., AND FOSTER, T.: AM. PRACT. & DIGEST. TREAT. 7:1992, 1956. 2. SUCKLE, E.: GERIATRICS 11:509, 1956. 3. FINCH, W. J.: J. OKLAHOMA M.A. 50:259, 1957. 4. TERMAN, L. A.: ILLINOIS M. J. 3:67, 1957. 5. GIFFORD, R. W.: J. ARKANSAS M. SOC. 55:31, 1958. 6. FORD, R. V., AND MOYER, J. H.: POSTGRAD. MED. 23:41, 1958.

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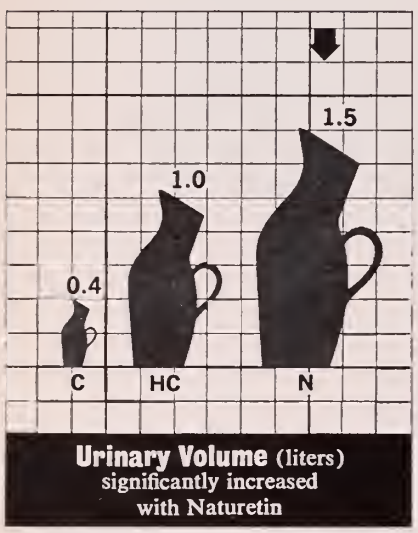
more closely approaches the ideal diuretic

Naturētin

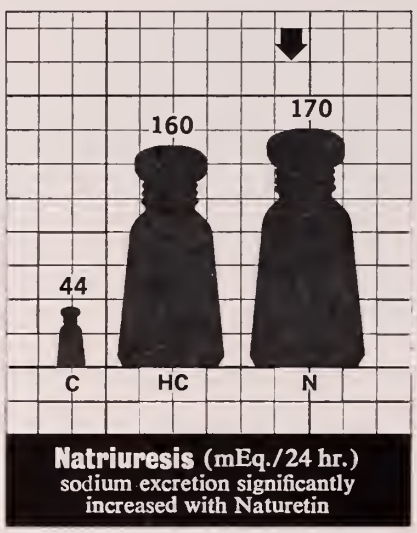
Squibb Benzydroflumethiazide

"When compared to other members of this heterocyclic group of compounds, this drug [NATURETIN] shows a significantly increased natriuresis and decreased loss of potassium and bicarbonate. In this respect it more closely approaches a natural or 'ideal diuretic.' It is effective upon continuous administration and causes no significant serum biochemical changes. It is effective in a wide variety of edematous and hypertensive states and represents a significant advance in diuretic therapy." *Ford, R.V.: Pharmacological observations on a more potent benzothiadiazine diuretic; accepted for publication by the American Heart Journal.*

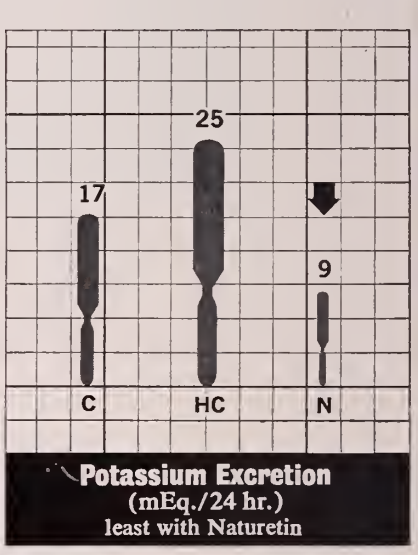
Comparison of electrolyte excretion pattern for the 24 hours following typical doses of chlorothiazide, hydrochlorothiazide, and Naturetin[1]



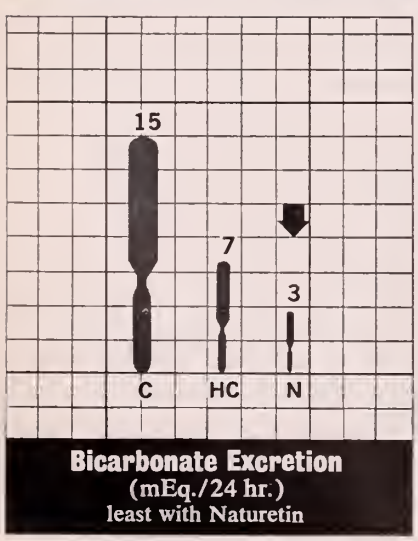
Urinary Volume (liters) significantly increased with Naturetin



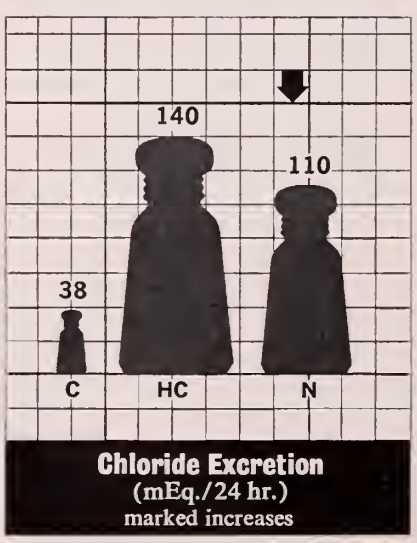
Natriuresis (mEq./24 hr.) sodium excretion significantly increased with Naturetin



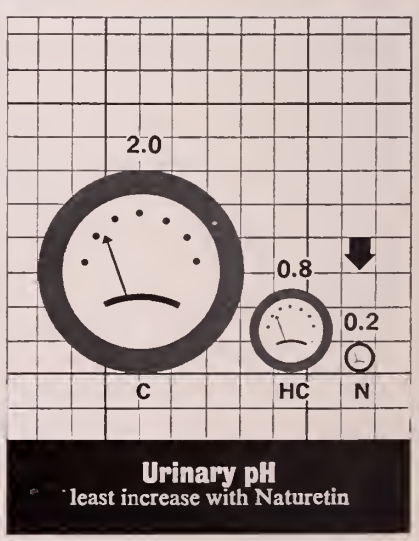
Potassium Excretion (mEq./24 hr.) least with Naturetin



Bicarbonate Excretion (mEq./24 hr.) least with Naturetin



Chloride Excretion (mEq./24 hr.) marked increases



Urinary pH least increase with Naturetin

Typical Doses: Chlorothiazide—1,000 mg.; Hydrochlorothiazide—50 mg.; Naturetin (Benzydroflumethiazide)—5 mg.

1. Adapted from: Ford, R. V., Squibb Clin. Res. Notes 2:1 (Dec.) 1959.

A single 5 mg. tablet once a day provides all these advantages[2]

- prolonged action — in excess of 18 hours
- convenient once-a-day dosage
- low daily dosage — more economical for the patient
- no significant alteration in normal electrolyte excretion pattern
- repetitively effective as a diuretic and antihypertensive
- greater potency mg. for mg.—more than 100 times as potent as chlorothiazide
- potency maintained with continued administration
- low toxicity — few side effects — low salt diets not necessary
- comparative studies with chlorothiazide, hydrochlorothiazide, and Naturetin disclose that smallest doses of Naturetin produce greater weight loss per day
- in hypertension, Naturetin, alone or in combination with other antihypertensives, produces significant decreases in mean blood pressure and other favorable clinical effects
- purpura and agranulocytosis not observed
- allergic reactions rarely observed

[2]Reports (1959) to the Squibb Institute for Medical Research.

Naturētin —*Indications:* in control of edema when diuresis is required, in congestive heart failure, in the premenstrual syndrome, nephrosis and nephritis, cirrhosis with ascites, edema induced by drugs (certain steroids); in the management of hypertension, used alone, combined with Raudixin (Squibb Rauwolfia Serpentina Whole Root), or with other antihypertensive drugs, such as ganglionic blocking agents.

Contraindications: none, except in complete renal shutdown.

Precautions: when Naturetin is added to an antihypertensive regimen including hydralazine, veratrum, and/or ganglionic blocking agents, immediate reduction must be made in the dosage for all preparations; the dosage for ganglionic blocking agents must be decreased by 50% to avoid a precipitous drop in blood pressure. This also applies if these hypotensive drugs are added to an established Naturetin regimen . . . in hypochloremic alkalosis with or without hypokalemia . . . in cirrhotic patients or those on digitalis therapy when reductions in serum potassium are noted . . . in diabetic patients or those predisposed to diabetes . . . when increased uric acid concentrations are noted . . . when signs—leg or abdominal cramps, pruritus, paresthesia, rash—suggestive of hypersensitivity, are noted.

Naturētin —*Dosage: in edema,* average dose, 5 mg., once daily, preferably in the morning; to initiate therapy, up to 20 mg., once daily or in divided doses; for maintenance, 2.5 to 5.0 mg., daily in a single dose. *In hypertension:* suggested initial dose, 5 to 20 mg. daily; for maintenance, 2.5 to 15 mg. daily, depending on the individual response of the patient. When Naturetin is added to an antihypertensive regimen with other agents, lower maintenance doses of each drug should be used.

Naturētin —*Supplied:* tablets of 2.5 mg. and 5 mg. (scored).

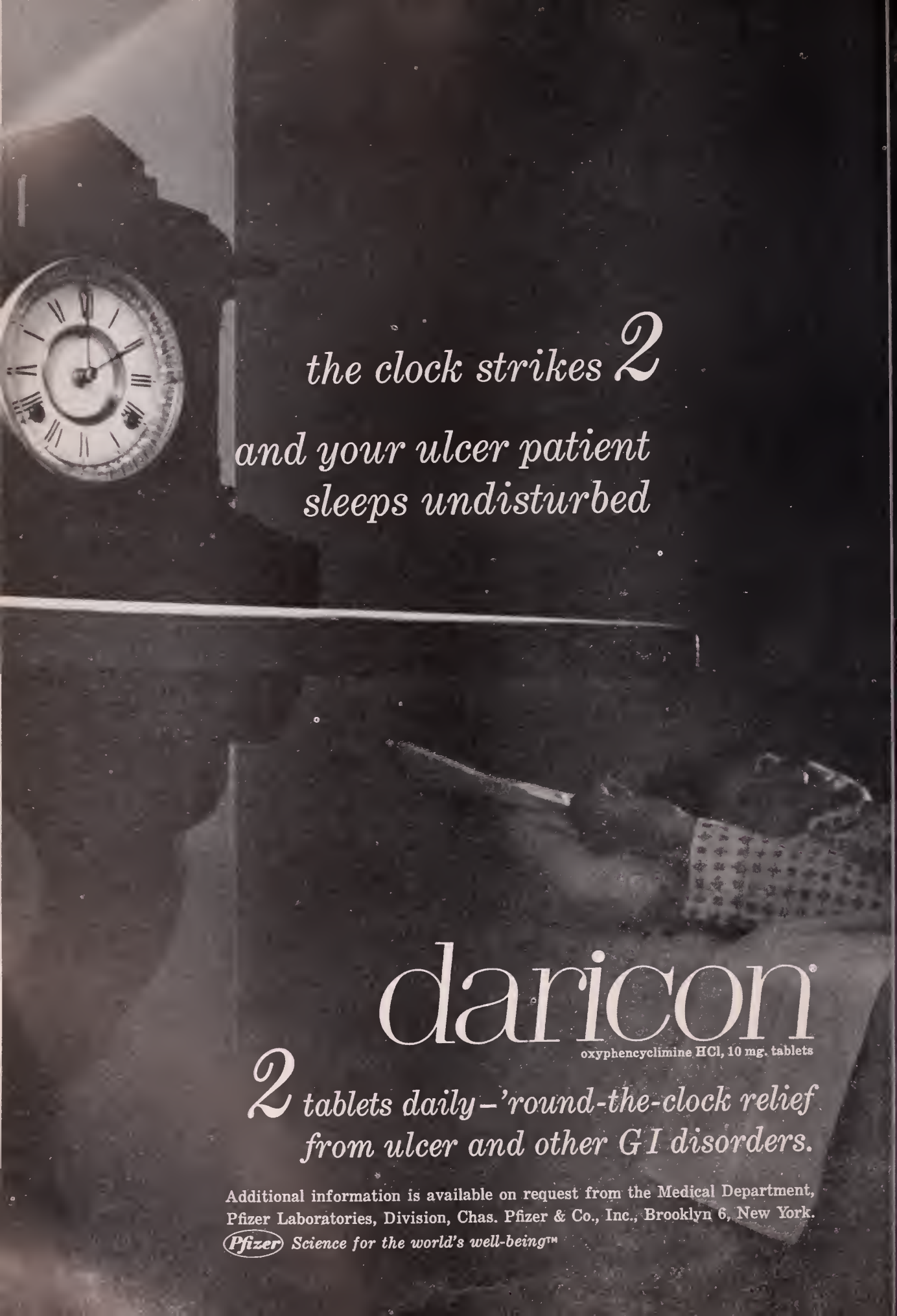
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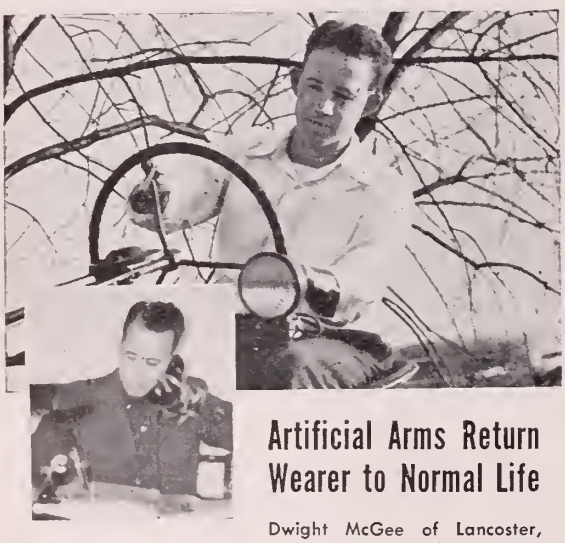
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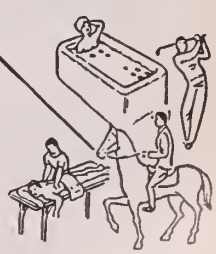
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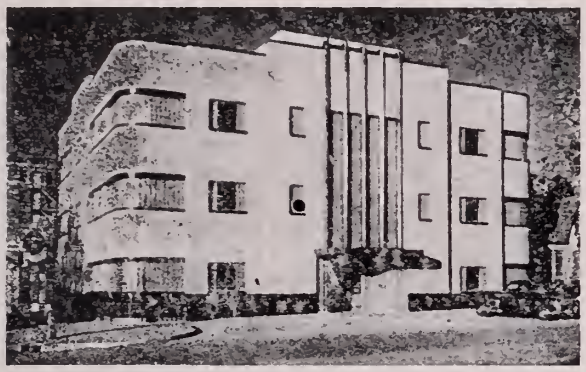
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Index to Advertisers

Abbott Laboratories 198, xxiv
Ames Company, Inc. inside back
Arlington Hotel lxi
Ayerst Laboratories xxv
Beverly Hills Clinic lxxi
Bone and Joint Hospital lxv
Bristol Laboratories, Inc. xxxi, xlvi-xlix
Burroughs-Wellcome & Company, Inc. insert
Ciba xx, xxix
Coca Cola Bottlers lxi
Corn Products Refining Co. xviii and xix
Coyne Campbell Sanitarium lxv
Credit Service lxi
Endo Laboratories li
First National Bank & Trust Co. xxxv
First Texas Pharmaceuticals, Inc. liv
Geigy xvii, xxxiii
General Electric xv
Glenbrook Labs xvi
Goldfain Laboratory lxvi
J. E. Hanger lx
Drs. Lain, Lamb and Jones lxvi
Lederle Laboratories xii, 188 and 189, xxx, xxxvii, insert
Liberty National Bank & Trust Co. xxvii
Eli Lilly & Company xiv
Medical Arts Laboratory lxiv
S. E. Massengill Company xxi
Melton Co., Inc. lxii
Merck Sharp and Dohme, Inc. iv, xxxviii, xliv
McAlester Clinic lxvii
Midwest Surgical Supply Co., Inc. lxx
Neurological Hospital lxxi
North American Company lix
Oklahoma Allergy Clinic lxvii
Osler Radioisotope Laboratory lxx
Parke Davis and Company inside front and iii
Pfizer Laboratories xxxii, xl, l, lviii
Physicians Casualty & Health Assn. lxii
Physicians Laboratory lxx
A. H. Robins Co., Inc. vi, xxxiv
Rock Haven Hospital lxxi
Roerig x, xxii, xlii and xliii
St. Paul Companies lxiii
Sardeau, Inc. xxiii
G. D. Searle & Company 159
Schering Corporation vii, xiii, lx
Seven-Up Bottlers lxii
Smith-Dorsey xxxix, xlv, lv
Smith, Kline & French Laboratories outside back
Springer Clinic lxviii
E. R. Squibb and Sons xi, xxvi, xli, lii, lvi and lvii
Stout Clinical Laboratory lxviii
Sugg Clinic lxix
Terrell's Laboratories lxx
Timberlawn Psychiatric Center lxiii
Tulsa Clinic lxix
The Upjohn Company viii and ix
Wallace Laboratories insert, liii
Winthrop Laboratories insert, xxviii, xxxvi

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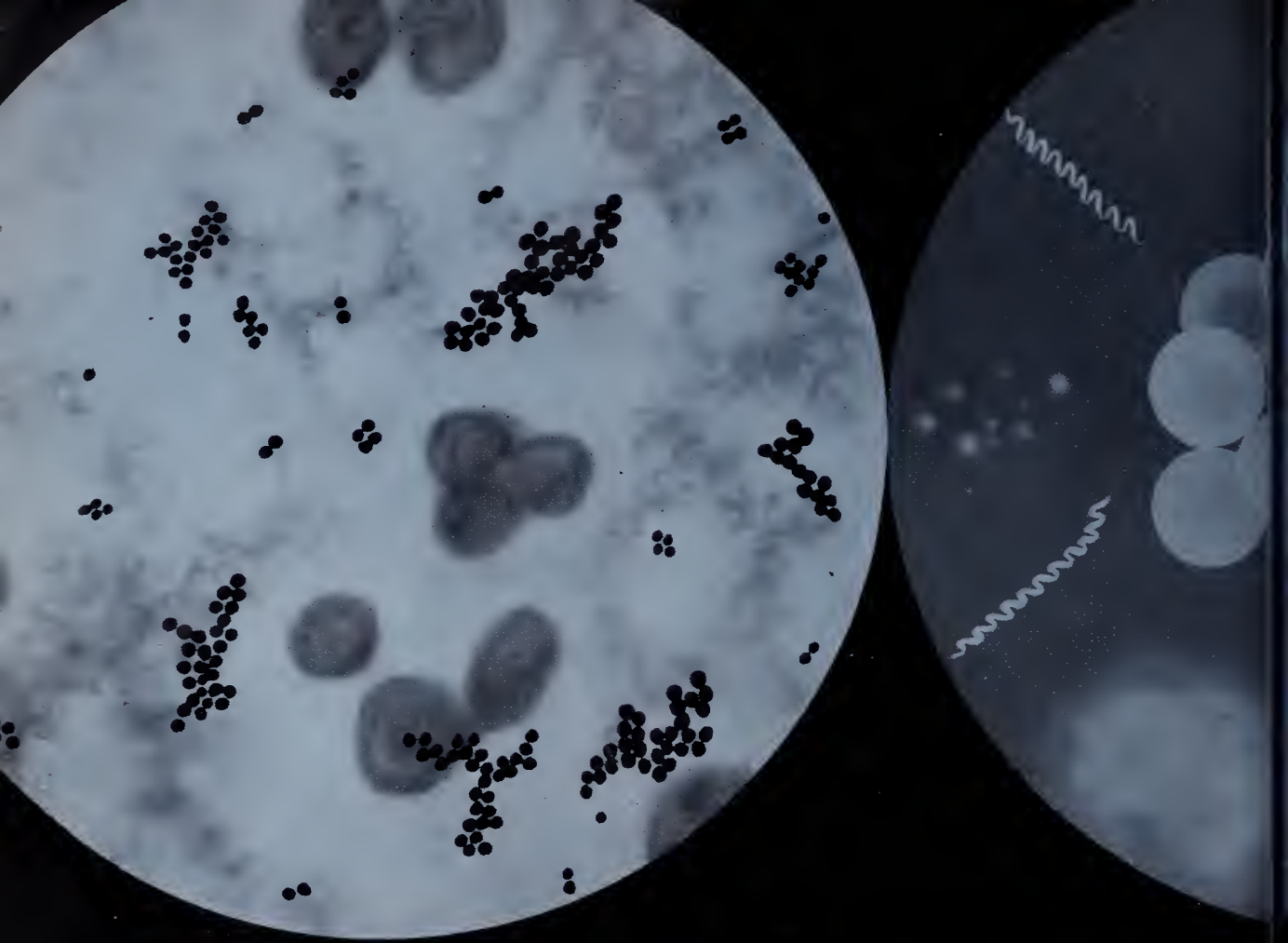
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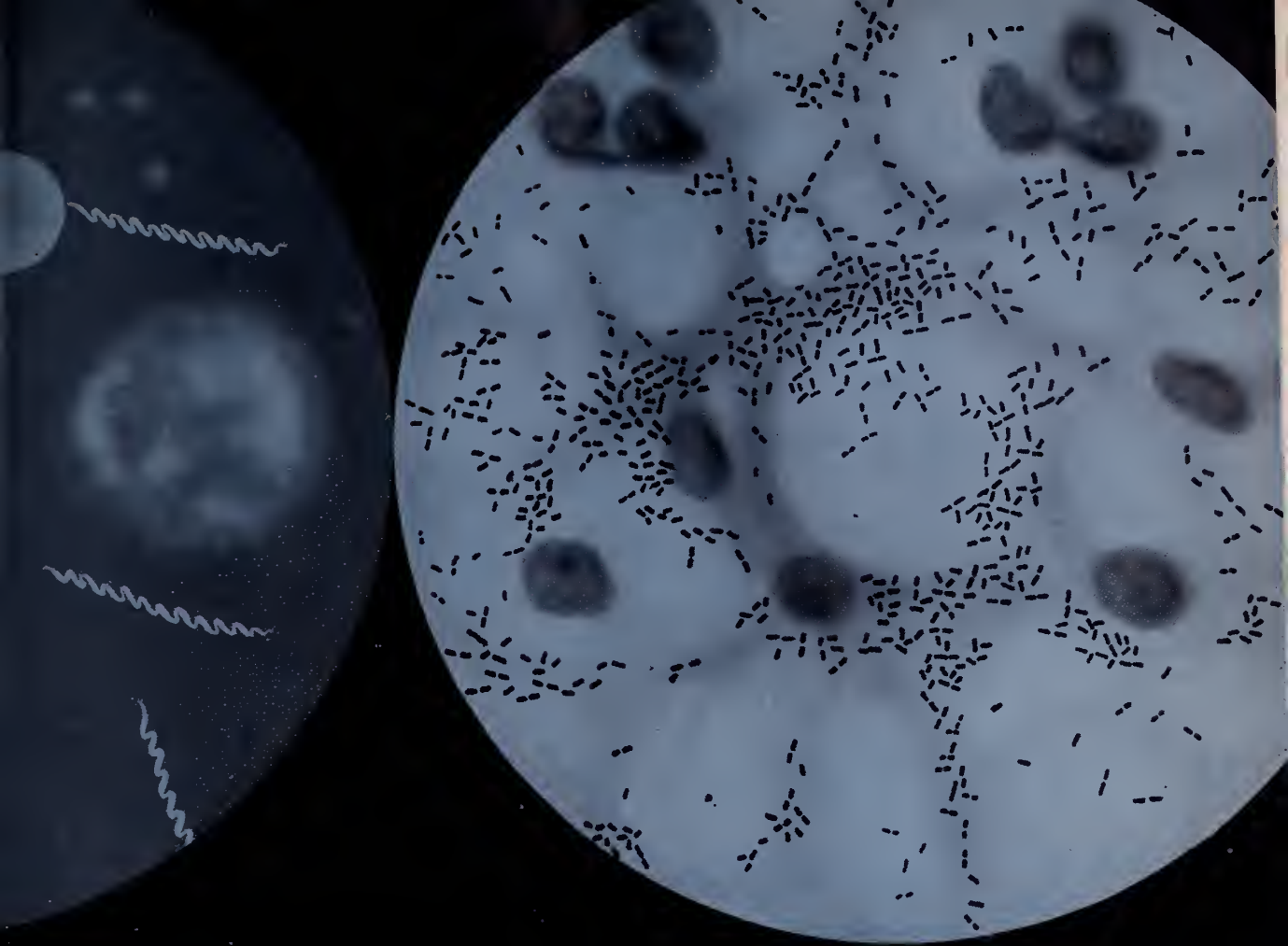
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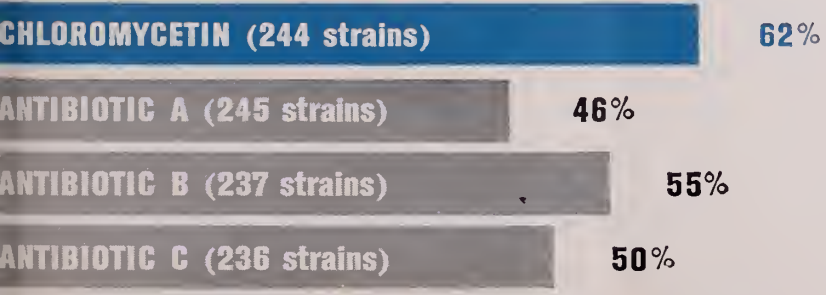
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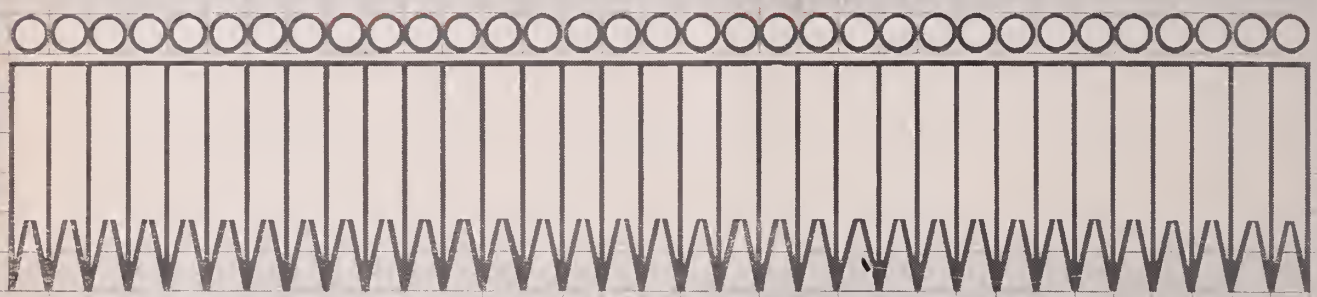
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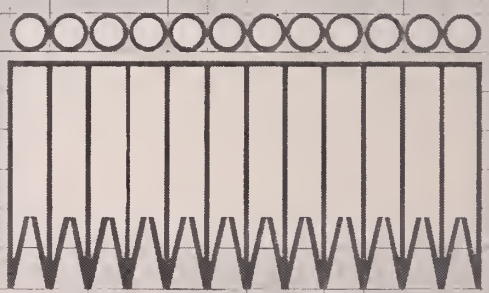
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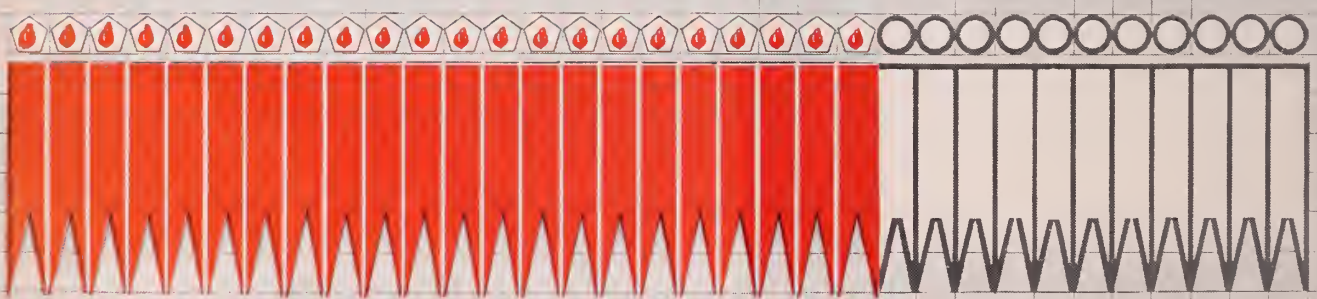
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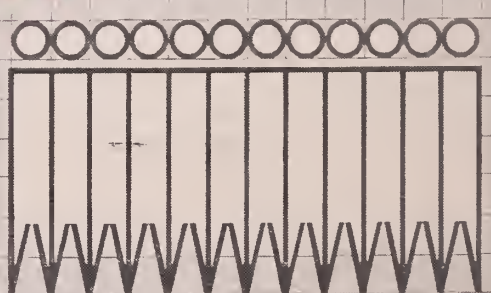
22 were successfully
treated with **Decadron**[1,2]

1. Boland, E. W., and Headley, N. E.: Paper read before the Am. Rheum. Assoc., San Francisco, Calif., June 21, 1958.
2. Bunim, J. J., et al.: Paper read before the Am. Rheum. Assoc., San Francisco, Calif., June 21, 1958.

*Cortisone, prednisone and prednisolone.

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APRIL 1960
Volume 53 Number 4

Journal
OF THE OKLAHOMA STATE MEDICAL ASSOCIATION

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CONTENTS

EDITORIALS

The Editor's Resignation 199
President's Letter 214

MEDICAL CENTER

Message from the Dean 204
Human Growth Hormone, *Doman K. Keele, M.D.* . . . 205
Case Report: Lead Intoxication, *E. Eugene Smith, M.D. and Edward T. Shirley, M.D.* 209
Abstracts 212
Faculty News 213

SPECIAL ARTICLE

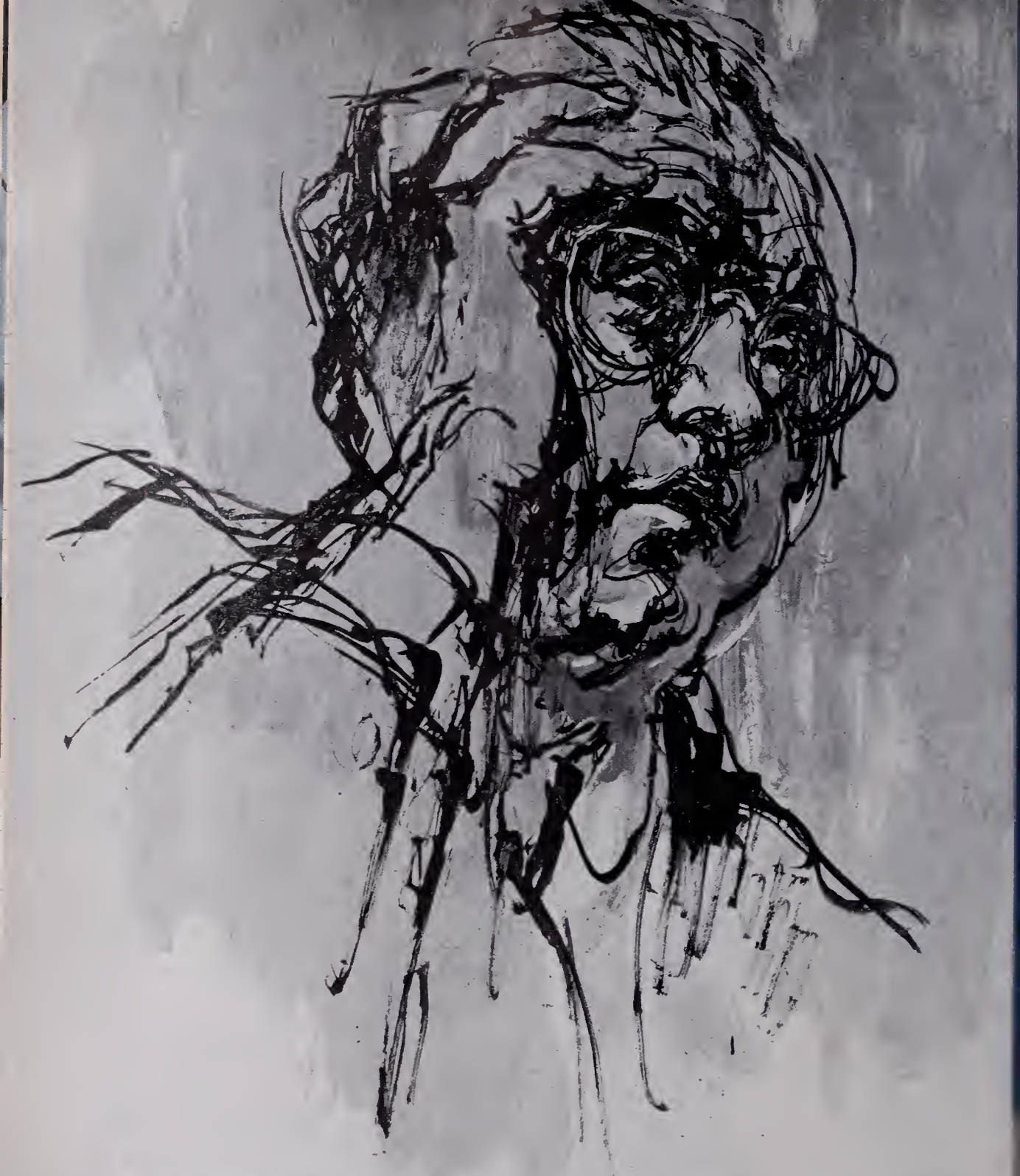
Factors Relating To Physician Distribution in Oklahoma, 1950-1959, *Robert C. Lowe, M.D., James A. Hagans, M.D. and Pearl Fisher, Ph.D.* 216

ANNUAL MEETING

Annual Meeting Preview 219-244
Officers and Councilors 220
Committees 221
Digest of Events 222
Guest Speakers 224
Scientific Program 228
Exhibits 230
Entertainment 234
Delegates 236
Related Meetings 238
Auxiliary Meetings 240
Medical Assistants Program 243

NEWS

Forand Threat Eases; More Trouble Ahead 245
Miami Beach Offers Ideal Climate, Diverse Activities for AMA Meeting 246
Heart Patient Course and Special Seminars Planned . 247
Tulsans Hear AMA Leader 247
Preceptors Listed for Medical School 252
Steve Stahl Honored 252
Coming Meetings 255
Five More Courses Offered by A.C.P. 254
Twenty-five Years Ago 256
Physician Placement 257
Miscellaneous Ads xv
Index to Advertisers lxxviii



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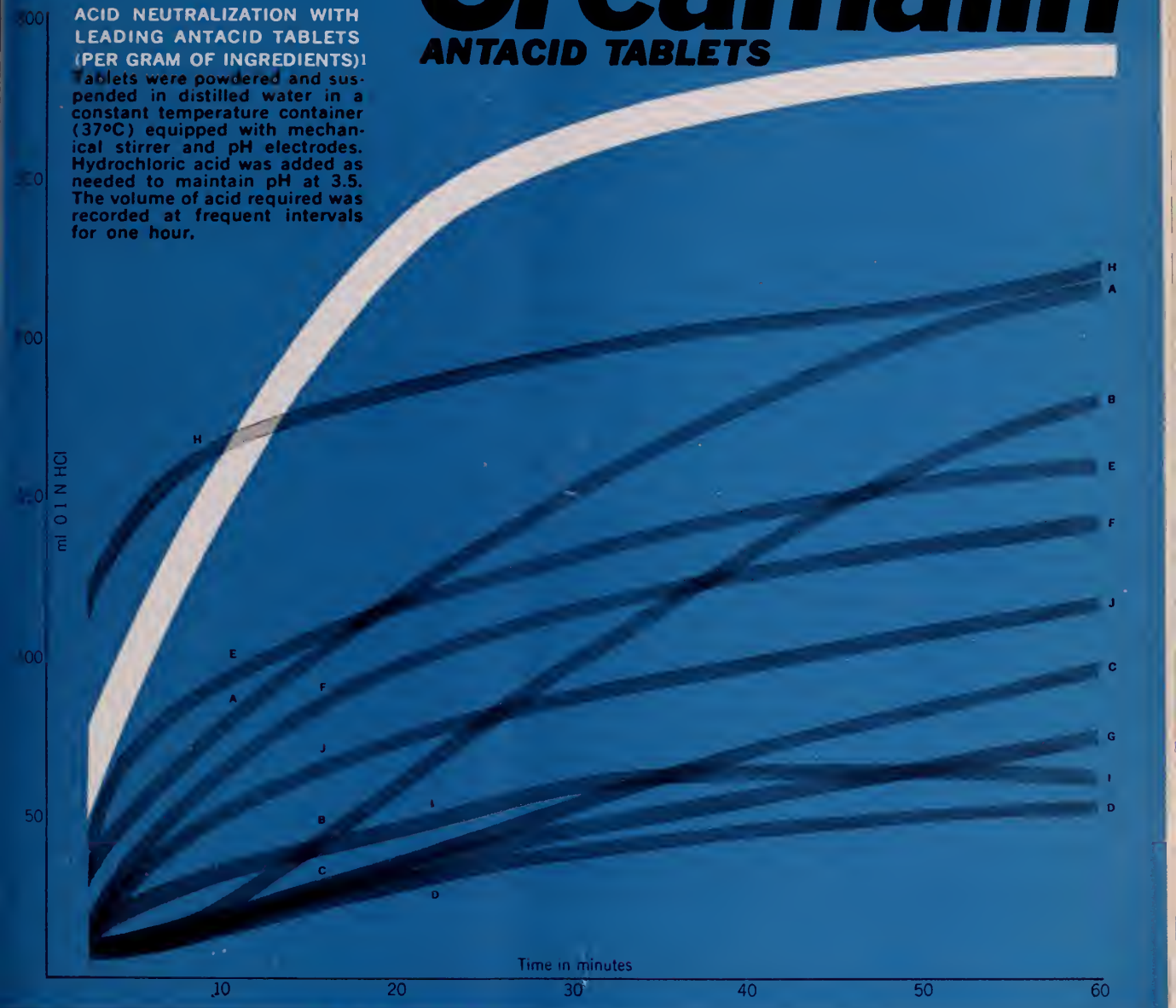
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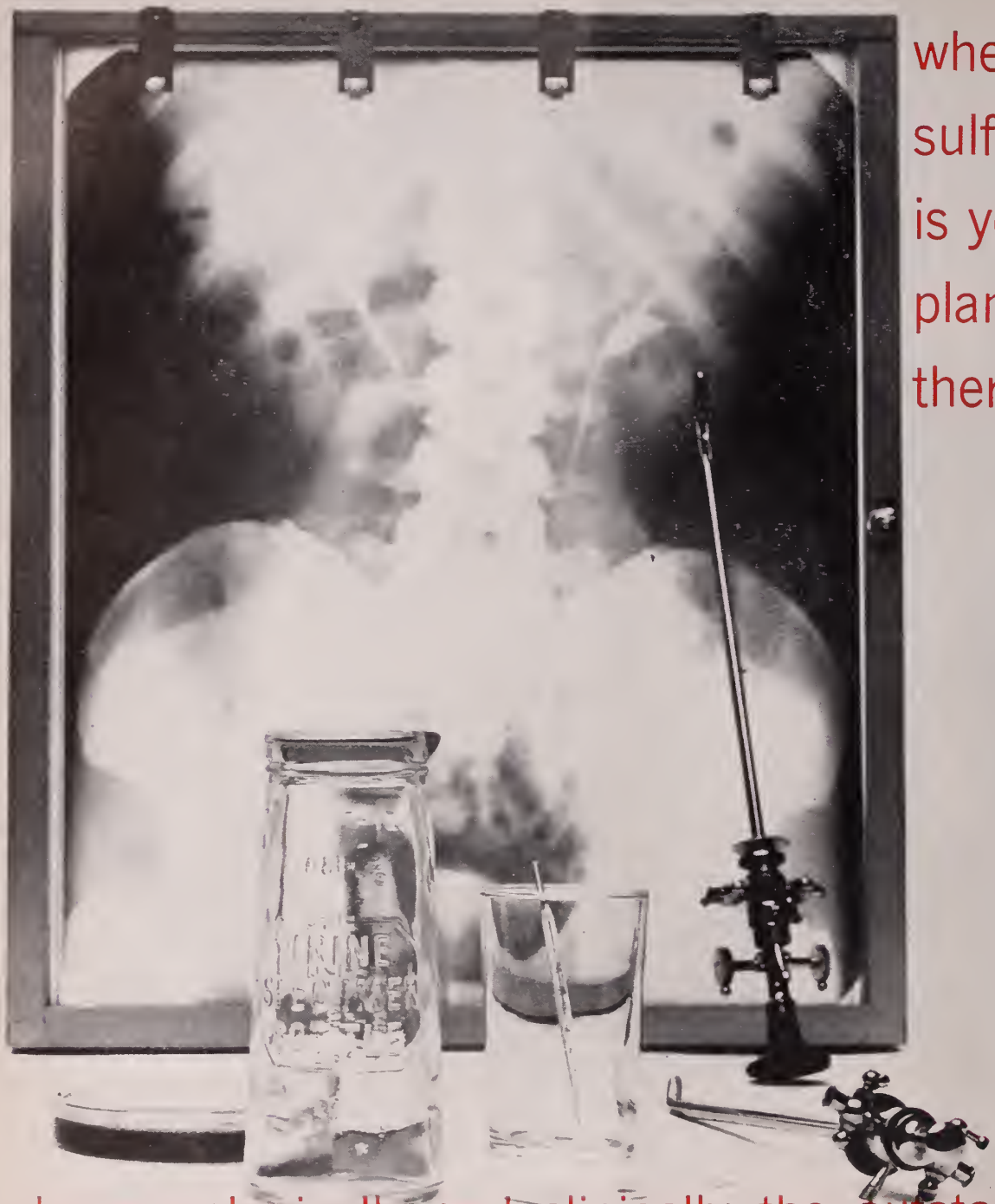
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1. Hinkel, E. T., Jr.; Fisher, M. P., and Tainter, M. L.: *J. Am. Pharm. A. (Scient. Ed.)* 48:380, July, 1959. 2. Hinkel, E. T., Jr.; Fisher, M. P., and Tainter, M. L.: *J. Am. Pharm. A. (Scient. Ed.)* 48:384, July, 1959.

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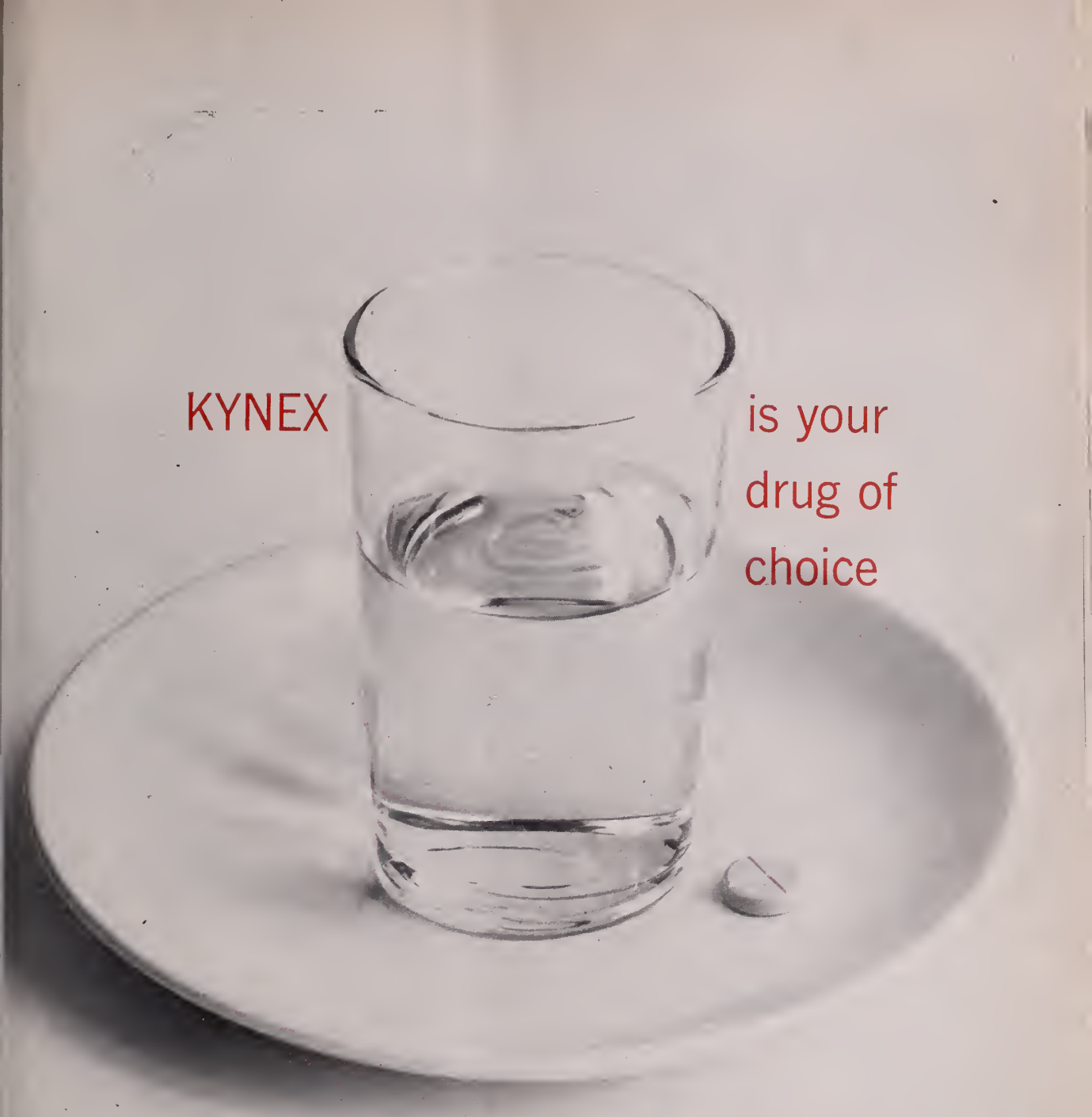
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1. Boger, W. P.; Strickland, C. S., and Gylfe, J. M.: Antibiotic Med. & Clin. Ther. 3:378, (Nov.) 1956. 2. Boger, W. P.: Antibiotics Annual 1958-1959, New York, Medical Encyclopedia, Inc., 1959, p. 48. 3. Sheth, U. K.; Kulkarni, B. S., and Kamath, P. G.: Antibiotic Med. & Clin. Ther. 5:604 (Oct.) 1958. 4. Vinnicombe, J.: Ibid. 5:474 (July) 1958. 5. Anderson, P. C., and Wissinger, H. A.: U. S. Armed Forces M. J. 10:1051 (Sept.) 1959. 6. Roepke, R. R.; Maren, T. H., and Mayer, E.: Ann. New York Acad. Sc. 60:457 (Oct.) 1957.



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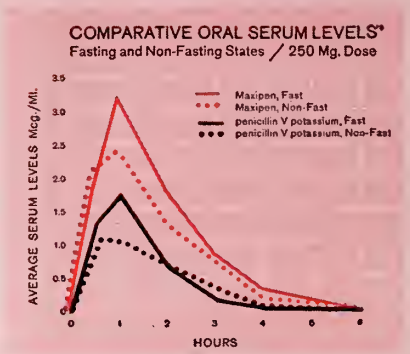
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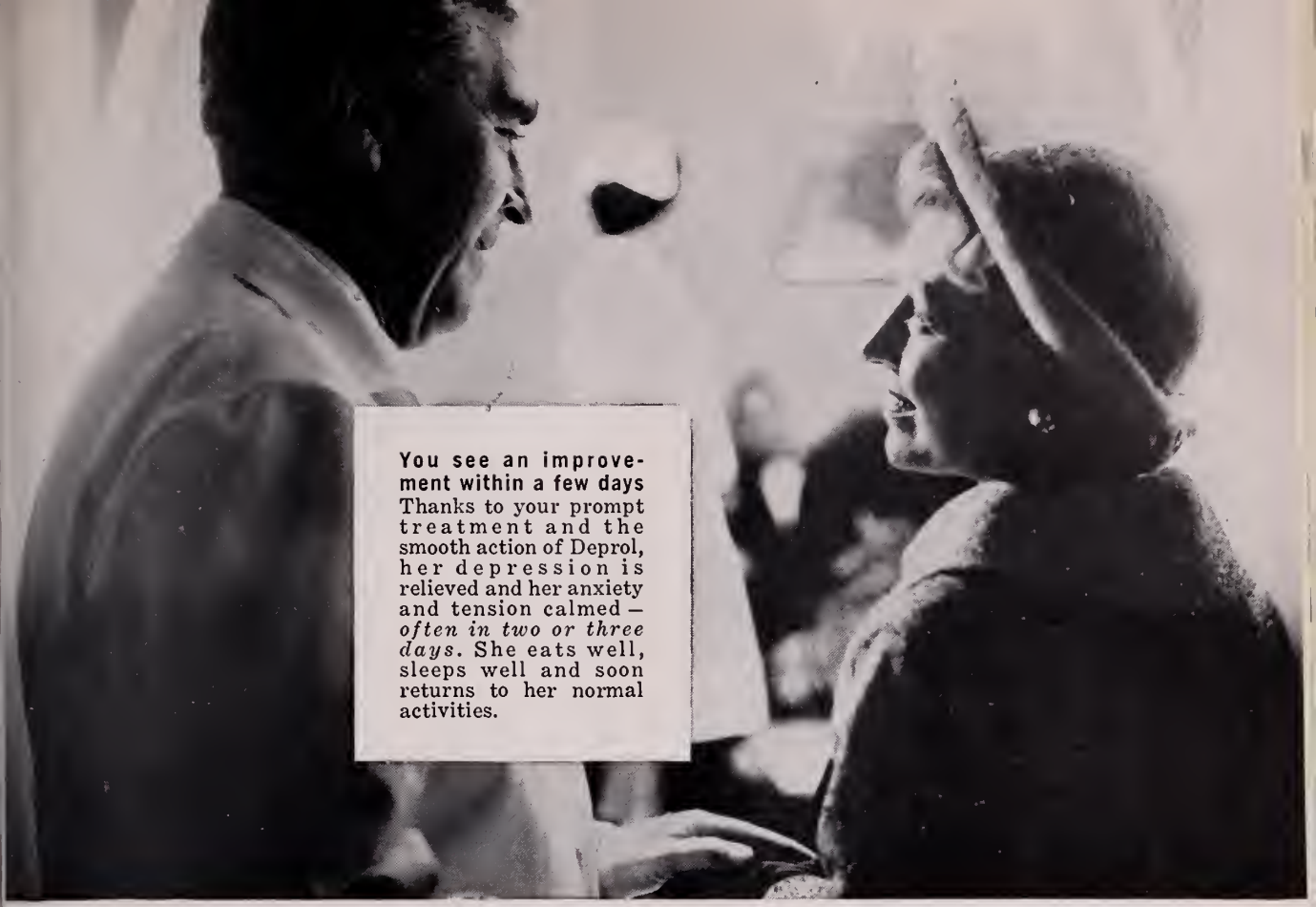


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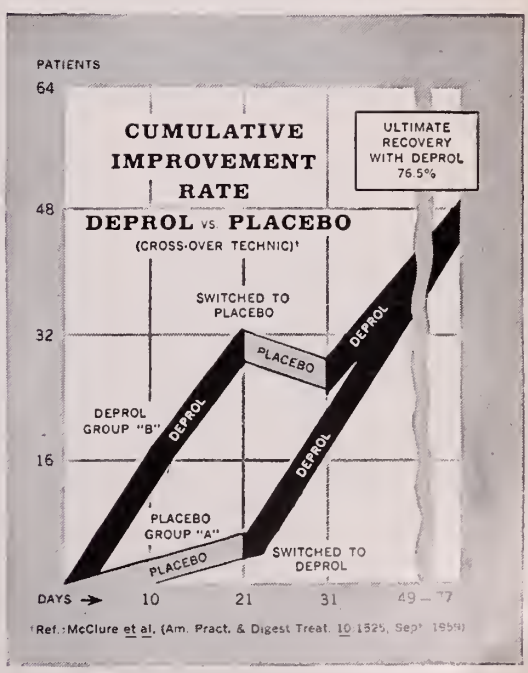
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[1]Ref.: McClure et al. (Am. Pract. & Digest Treat. 10:1525, Sept. 1959)

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1. Cook, J.E., Briggs, G.W., and Hindley, F.W.: Chronic Amebiasis and the Need for a Diagnostic Profile, Am. Pract. and Dig. of Treat. *6*:1821 (Dec., 1955).

2. Rinehart, R.E., and Marcus, H.: Incidence of Amebiasis in Healthy Individuals, Clinic Patients and Those with Rheumatoid Arthritis, Northwest Med., *54*:708 (July, 1955).

3. Webster, B.H.: Amebiasis, a Disease of Multiple Manifestations, Am. Pract. and Dig. of Treat. *9*:897 (June, 1958).

*U.S. Pat. No. 2,864,745

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1. Smith, I. M., and Soderstrom, W. H.: J. A. M. A., *170*:184 (May 9), 1959.

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Editorial

February 29, 1960

Alfred T. Baker, M.D.
Durant, Oklahoma

Dear Doctor Baker:

For personal reasons it is necessary for me to give up the Editorship of The Journal. *My association with the entire staff at the Association's headquarters has been so pleasant that it is with great reluctance I ask to be replaced. Mr. Graham has at all times been earnestly interested in* The Journal *and Journal problems and has not only been helpful by intent but in many ways of which he was unaware. Mr. Blair has ably taken on that portion of* The Journal *which is not part of the scientific section. Mrs. Martin has moulded it all together into one of the best State Medical Journals presently published. Mr. Nance of the Transcript Company, our publishers, has been interested and helpful in making needed changes in format and his advice has been relied on in many other ways. To all of them I am most grateful for their specific part and to the others of the headquarters staff for their general cooperative spirit of which the Association should be proud.*

Since continuity of concern about the affairs and future of the Association at an administrative level by members is not predictable because of the Association's democratic nature, it seems fitting to set out a few impressions gained from the vantage point of nearly six years as Editor. This will be done in the editorial section of The Journal.

Be assured of my continued interest in the Association and in The Journal. *The rapport that has developed between* The Journal *and the University's Medical Center should continue to be cherished, for it has been mutually beneficial to an incalculable degree.*

Sincerely
Ben H. Nicholson, M.D.

On Organized Medicine

This term to me means the American Medical Association and its constituent medical societies in action. Organized medicine does a beautiful job in matters with which it should be concerned which can be examined, turned over, viewed from all angles and critically evaluated objectively. In these areas policies can be developed which are consistent, which are progressive and which have the best interests of all concerned in mind. These policies are altered and redirected only when new truths are found which makes changes mandatory.

Policies and direction are less well defined, less consistent and much less predictable when matters of concern are, because of their nature, considered subjectively. It is difficult for an individual or an organization, representing any line of endeavor, to change from ways and means to which they have become accustomed. American culture and way of life since the Second World War has, by no means, been static. Medicine itself has seen many phenomenal changes. The integration of the practice of medicine into economic, social and cultural change is far more a subjective problem of the physician than an objective problem of his organization. This makes it impossible for satisfactory solutions to be solved at the level

of a national organization. Sound thinking and planning for fitting the practice of medicine into change must be made at the local level.

If we are to keep medicine free, and we must, we must make our system encompass those changes within its structure. We cannot survive if all we do is resist change and secure the services of public relations people to extol our virtues.

On Public Medicine

As used here the term means *a sharing of the responsibility between the public and the medical profession for the medical care of those people who cannot for one reason or another see to their own.* Such sharing has long been accepted as both necessary and desirable in the care of many people who have tuberculosis or who have mental disease. When the only reason is an economic one and the disease less prolonged and less devastating to the family, the justification for public sharing is less clear cut.

The attitude of the association toward public medicine has been commented on editorially many times.[1, 2, 3, 4, 5, 6, 7] There is little need to belabor it further here. The idea of public medicine is so abhorrent that our people were orginally unwilling to recognize the need for any extension of it at all. After the D.P.W. Medical Care program went into effect and the AMA asked us to consider the over-65 group on Social Security, it seemed reasonable to hope that we would not only recognize shades of black but shades of gray as well; and would set about the task of solving the medico-economic problems of those people in the gray area in such a way that they would not be included in the group to be taken care of as public charges (state medicine).

We either recognized no problem or recognizing it refused to accept responsibility for its solution. If the public thinks there is a problem, and I suspect that it increasingly does, attempts at a solution, since we offer none tangible enough to be worth mentioning, will keep on originating in the Congress until, eventually, one will be adopted. It is too bad for, as the cost of total medical care goes up, the gray area will be extended and we will be up to our umbilici in state medicine.

On Blue Shield

As a result of the success of Blue Cross in helping people budget for the cost of their hospital care—we, the association, set up Blue Shield[8, 9] to do the same for medical care. Somewhere between 1945 and now the continuity of purpose was lost, and we began to rationalize that since the insurance industry is private enterprise and since we want to continue as a private enterprise, it would not be consistent for us to go all out for our own creation to do the job for us. Arguing the right and wrong of this has so far availed nothing,[10] but this attitude has deprived us of a useful tool. We rejected a chance to use it in the D.P.W. program which would have given us invaluable experience in a positive way for keeping the gray areas out of political medicine, and we are now once again fighting a negative delaying action against a new Forand-type of legislation. Infants need only be burned once to avoid fire.

On the Family Doctor

An effort has been made to recreate the image of the family doctor of 50 years ago and place him on a pedestal in 1960—a sort of parachronism or anachronism in reverse. I know nothing of the public relations value of this sort of distortion of truth, but the fact that it is such a distortion makes it unwise. In analyzing this, it appears that the state association is largely responsible. This is a delicate subject and I hope if it cannot be handled delicately, it will, at least, not be handled indelicately. We must search for the truth—bear me out.

The general practitioner or family doctor today is not an entity. He differs in only one respect from any other physician or so-called specialist. Whereas the specialist devotes himself to a particular field and has an academic interest in all medicine, the generalist has a particular field in which he is most interested and does his best work but

by circumstance his interest in other fields is not just academic but is a working, practical one. A failure to recognize this has been a serious error on the part of the association, in this way:

With the development of the Board man and the specialty societies, interest in the section meetings of the association lagged and it was considered expedient to abandon them in favor of inviting many big names to speak at the annual meeting. The generalist with a special interest was then denied the association, the encouragement and the help that formerly came when he met with men specializing in his particular field of interest at a section meeting. Although the family doctor has always been the back bone of the state association, he is now pretty much out in the cold for his previous colleagues, the specialists, have ridden off, each in his own direction.

The wisdom of abandoning the section meetings is not questioned but the substitute is. The state association,[11] at this point, should have gone out of the scientific meeting business and should have requested the specialty groups to have their meetings under the umbrella of the annual meeting with any member of the association invited to attend and enter into the discussion. This would have brought the specialty groups and the Academy of General Practice back under the wing of the parent organization and would have provided immeasurably better scientific programs. *The Journal* would have again had the annual meeting as a source of supply for scientific material—almost no material comes from the annual meeting now, for only a very few members of the association appear on the program.

An effort to begin this change was made in 1957 when the Oklahoma Chapters of the American Academies of Pediatrics and of Obstetrics and Gynecology petitioned the Council for this sort of arrangement for their groups. The petition was denied. The Radiologists, the Rheumatologists, the Orthopedists, the Psychiatrists and perhaps others have had their meetings on Sunday before the state association's annual meeting and gone home.

On the Medical School and Medical Education

I appeared as a member of the Dean's faculty at an inquisition of the medical school by the state chapter of the Academy of General Practice held at the association's headquarters under the auspices of the association's Medical School Liaison Committee. An unofficial, but intended, news release reached the public press from the Academy's state meeting castigating the school of medicine for being ineffectual in training men for general practice. That an attempt should be made to discredit the school by the men it sired is incredible. Maybe the word *liaison* is not understood or the committee is inappropriately named. The contemplation of the full significance of this is so depressing to those who believe in the mutual support of men going in the same direction and with the same purpose, that we should put it out of mind and turn to a consideration of the medical school and medical education[12] in 1960, not 1910.

The medical school has no right to force any student into any particular field of endeavor. Medical Education today is an educational process not an exercise in vocational training. The change-over began seriously with the Flexner report in 1910. Oklahoma was late in beginning to effect it; consequently many of our association's members may not be aware of the change in approach to Medical Education. The vocational training necessary to qualify one to practice begins after graduation. Some of it naturally occurs during the undergraduate period but it is incidental, not purposeful.

As a matter of fact, the preclinical subjects, such as anatomy, biochemistry, microbiology and physiology should be taught for themselves alone and not just with an eye toward clinical medicine as a sort of necessary, but boring step in that direction. These courses should be attractive enough and stimulating enough that an occasional student will find in one of them his life's work. One can't, out of one side of the mouth resent the importation of outside teachers and out of the other, deplore the use of the facilities, the time, the personnel and the

research necessary to stimulate some of our own students to enter the many fields of academic medicine. It just doesn't make sense anyway one views it. If these concepts are true and desirable, for the school to be preoccupied with the training of students to do general practice would be ridiculous. I would agree, however, that the school should extend its interest in students beyond graduation and should concern itself with the quality of training programs in the Oklahoma hospitals designed to lead to a life of general practice.

On the Preceptorship

The university and the students have been fortunate in the character and quality of the preceptors on the faculty. These men have worked hard and patiently to give the preceptee the training and experience desired for them. This program was inaugurated to encourage students to ultimately go into general practice in small communities. The unwarranted blast at the medical school by the Academy of General Practice would lead one to believe that it has not been very effective in accomplishing the purpose for which it was intended. If this is true, then it should no longer be compulsory and some of the students who earnestly desire to do so, should, with the faculty's permission, spend this time in other pursuits. For the real need of the people of Oklahoma, there is no more reason to require students to take a preceptorship to guide them into general practice than there is to require them to take a preceptorship in anatomy, in pathology, in clinical pathology, in radiology and in otolaryngology to guide them into these fields. The yield for general practice should be higher if the preceptee group were undiluted and undisturbed by boys who are not interested and who may resent being required to take the course.

On the Crippled Children's Program

There is a move under way to secure from the House of Delegates, a request for the Department of Public Welfare to pay members of the association for the medical care of indigent children. The reason for this attempt to reverse the association's stand of 20-odd years is not clear unless the succulence of the public tit discovered in the D.P.W.'s old age medical care program was found not to be so distasteful after all.

In response to such a request if I were the state, I would say, "I agree with you, you should be paid for the service you give these children and an effort will be made to arrange it. There are, however, certain facets of this of which you should be aware. I have assumed that adults have enough faculties to guide their own destiny and I have not been concerned about where and from whom they got their care as long as those who provided it were qualified by state law to do so. Children are under my custody just as they would be under a parent's. I, too, desire that they grow up into useful citizens with as much of their potential physical, mental and emotional equipment intact as possible.

"Under the present method of operation, children have, for the most part, been protected against ill-advised and incompetent treatment by the fact that no money is involved; and indirectly by the fact that hospitals have been divided into categories for treatment, depending on the number and kind of ancillary services they offer and the qualifications of their staff members. Under your proposed plan, it will be necessary for me to stipulate what physicians can do what, and where they can do it.

"I realize that provisions for emergency care must be set out. I trust that your request will carry safeguards against their abuse and that I will be spared the odious task of imposing them."

If you were the state this would be your answer, too.

On Trade Unionism in Medicine

In the past few years there has developed a vein of trade unionism at its worst within a collateral medical society or two and to some extent within our own association. This adaptation takes its form in the conviction that the welfare of medicine comes first and the welfare of the public next. As individual physicians we still maintain the traditional philosophy that the welfare of our patients comes first. It should not be too difficult to maintain this philosophy collectively and

feel that to organized medicine the welfare of the public comes first. It is an infathomable paradox to the layman that organized medicine does not think and behave as does the doctor he knows.

The real difficulty, I suppose, lies in the fact that we individually can easily recognize medical indigency in the single person or a family but we collectively cannot believe that segments of the entire population can be. These truths seem self evident to me:

1. We, not government, are responsible for the medical care of the people of the United States. But, government, with its taxing authority, can assume this responsibility by forcing an economic mediation of our medical care through payment for it by government. Those who are naive enough to think that organized medicine can resist this should recall the little phrase on the Medicare claim just above their signatures:

"I certify that I am not an intern or resident: that this statement for services rendered (which were performed ☐ a. by me as the attending physician or ☐ b. performed by me as authorized by the attending physician) is true and correct: that payment due from the government under P.L. 569 has not been received, and that, except for the amount shown under item 26: (Check appropriate box below)

☐ a. I will accept the amount listed in the Dependent's Medical Care Program schedule of allowances or the amount shown in item 24, whichever is less, as full payment for the services described above.

☐ b. I will accept the amount shown in item 24 (which is supported by the attached special report as justification for services of unusual degree or for authorized services not covered in the schedule of allowances) upon approval and payment by the government as full payment for the services described in the attached report."

2. The group or segment over 65 on Social Security not now covered by the DPW's medical care program will, for the most part, be medically indigent if serious illness comes along. If this were not true I do not believe the American Medical Association would have asked us to come up with a positive program to solve the problem of their care.

3. The medical care of this group is our problem. Opportunity for adequate care they must have and they must have it by a plan that can be described and written out. Can we write it out or must congress write it out?

4. If we cannot, congress will. We do not want this to happen and I do not believe the people do.

5. Only we can prevent it and only by writing out the plan, not by publicity and political maneuvering except temporarily, year by year, and Congress by Congress.

6. We have traditionally cared for the poor without charge, cared for the average for an average charge, and for the well-to-do for often a greater fee. Our justification for the larger fee was the *no fee* for the poor. In other words we have been applying a tax system of our own which we deplore in government—we are reserving the right to soak the rich in order to pay for the poor. It can be called the sanctity of doctor-patient relationship or doctor-patient contract or by any other name—it still smells the same.

7. If the poor over 65 can pay for his own and is no longer to be cared for free, there can be no justification for maintaining a tax on the rich over 65 to provide for the poor man's care. This is the basis for and reasoning behind the service contract. It is unwillingness to accept the idea of a service contract that keeps us from offering a responsible plan that will prevent the government from taking over. When it happens, let's not blame the socialists, the pinks, the politicians, bureaucrats. Let us put the blame where it belongs, squarely on our own shoulders.—*B.H.N.*

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MEDICAL  CENTER

Message from the Dean

The Outpatient Clinics at University of Oklahoma Hospitals provide a unique opportunity for medical students to learn ambulatory medicine. The curriculum is so arranged that it is possible for a student to follow a patient in whatever clinic is appropriate for the patient's needs. This is achieved by assigning the student to each major clinic at least once weekly, and also by combining the functions of a screening clinic and an internal medicine clinic into one general medical clinic. Through this clinic all new patients are funneled and here each is assigned to a student. For patients under the age of 14 the general pediatrics clinic serves the same functions.

Under the direction of the clinical staff each patient is afforded as exhaustive and critical an evaluation as is suited to his medical needs. Treatment, further studies and an appropriate follow-up are formulated. The emphasis for follow-up of the patient and the responsibility for this is placed on the student but is closely supervised by the attending physician. This assumption of responsibility speeds the student in his intellectual growth so that he may assume properly the functions of the patient's physician. The attending physician, serving in the role of consultant, guides and directs this progress. In this environment, the medical student, the house officer, and the attending physician are all continually aware of their responsibilities as physicians for both patient care and mutual intellectual advancement.

It is our premise that the best patient care is the best teaching experience. It is important to recall that the University Hospitals are charged with providing patient material for the education of medical students and the training of the house staff and do not have sufficient facilities for the medical care of a great number of the state's medically indigent which interferes somewhat with our desire to be of service. In order to facilitate the selection of cases and to more efficiently schedule visits to clinics which meet only occasionally, all patients must be referred in by the practicing physician who has examined the patient and who incidentally by his referal indicates his agreement as to the patient's need and medical indigency.

There are conditions in which such a high order of study and analysis is required that these may not be available locally. In such situations we would like to be of service if physical space and personnel time permits.

Mark R Everett

Human growth hormone has been purified and found to have physiological and therapeutic effects in man. Methods are also available for its measurement in man.

HUMAN GROWTH HORMONE

DOMAN K. KEELE, M.D. *

Doman K. Keele, M.D., graduated from the University of Texas Southwestern Medical School in 1953. He is now Assistant Professor of Pediatrics at the University of Oklahoma Medical Center and is a member of Alpha Omega Alpha. He specializes in pediatric endocrinology.

BRILLIANT RESEARCH in the past three decades has shown the nature and action of animal growth hormone. In the past two to three years, great advances have also been made with human growth hormone; it has been highly purified and shown to be active in man. The purpose of this article is to review some of the physiological and clinical aspects of human growth hormone. Space does not permit a review of the massive amount of literature dealing with animal growth hormone; this literature will be referred to only when it relates to similar aspects of human growth hormone.

There are two methods of extracting growth hormone from human pituitary glands. One is the glacial acetic acid extraction method described by Raben.[1,2] The other method described by Li and Papkoff[3,4] involves extraction at an alkaline pH with calcium hydroxide. The yields of the two methods are comparable. Chemically, the hormone is a protein; it has the characteristic of a straight-chained polypeptide.[5,9] The molecule is similar to the monkey growth hormone molecule, but differs markedly from the beef growth hormone molecule which appears to be a branch-chained polypeptide. It is believed that this structural difference may account for the fact that the former two act physiologically in man whereas the latter one does not. Those interested in a more detailed description of the biochemical aspects of growth hormone should refer to the references.[1-14]

*From the department of Pediatrics and the Children's Memorial Hospital, University of Oklahoma Medical Center. Oklahoma City, Oklahoma.

Effects of Human Growth Hormone in Man

A large amount of evidence has accumulated in the literature in the last two decades showing the anabolic action of growth hormone in hypophysectomized animals. The anabolic action of growth hormone is characterized by an increase in skeletal size, weight, and protein content of the body. Chrondrogenesis and osteogenesis are stimulated resulting in skeletal enlargement. The muscles and such organs as the spleen, kidneys, stomach, intestines, and heart are increased in size. These changes are accompanied by a decrease of body fat content and an increase of protein and water content.[17] Balance studies have shown an increase of storage of nitrogen, phosphorus, potassium, calcium, and sodium.

The mechanism of the protein anabolic effect appears to be both an increase in the incorporation of amino acids into protein, and a decrease in the catabolism of protein. For an excellent review of this subject see Ketherer, et al.[18] It has been shown that insulin is necessary for the protein anabolic effect of growth hormone.[19] The role of insulin in this "synergistic" action is not known; it could be a primary action of in-

sulin on protein anabolism or a secondary one through carbohydrate metabolism.

Growth hormone also has been shown to have an effect on carbohydrate metabolism. It abolishes the insulin sensitivity that follows hypophysectomy, and decreases glucose uptake by muscle. A single injection of growth hormone causes hypoglycemia but repeated injections cause beta cell degeneration of the pancreas and if continued long enough cause permanent diabetes mellitus.[21] The hypoglycemic action of growth hormone may be due to the fact that it increases the rate of insulin secretion by the pancreas.[22] Once the diabetic state develops, the hypoglycemic action disappears, however.

As we have seen above, the injection of growth hormone decreases the fat content of the body of hypophysectomized animals. It also increases the fatty acid content of the liver.[16,17] It induces ketone formation and lowers the respiratory quotient. This is evidence that growth hormone causes the catabolism of fat stores. Greenbaum has adduced evidence that protein anabolism occurs only when fat catabolism is occurring.[16,17] This may mean that the former is dependent on the latter.

Recently a small amount of conclusive evidence has accumulated that shows that human growth hormone causes protein anabolism in man. This is characterized by skeletal enlargement[34] and storage of nitrogen, potassium, calcium and sodium.[20] There is both a positive calcium balance and an increased calcium excretion.[37,23] The blood urea nitrogen falls; this fact may reflect the anabolic action of growth hormone. Adrenal function appears to be unchanged except for increased excretion of aldosterone;[37] the explanation for this is not known.

Growth hormone has the same effect on carbohydrate and fat metabolism in man as in animals. Insulin sensitivity is decreased in hypopituitary patients; it also causes a rise of blood and urinary glucose as well as an increase in ketosis in hypophysectomized diabetic patients receiving insulin.[30,31] Plasma fatty acids rise after growth hormone injection; this suggests a fat mobilizing action of growth hormone.[24] This effect is abolished by food and glucose. Raben has suggested, on the basis of this evidence, that the action of growth hormone may occur during periods of fasting by making fat stores available through its fat mobilizing action.[24] This would fit with the hypothesis of Greenbaum that protein anabolism occurs only when fat catabolism occurs.[17]

The Physiologic Significance of Growth Hormone in Man

The responsiveness of the individual to the linear growth-promoting properties of growth hormone apparently begins after infancy and ends with the closure of the epiphyses at puberty. In congenital hypopituitarism, the infant usually has a normal birth weight and grows normally for the first two or three years of life. This seems to indicate that the child can grow normally during early life without growth hormone. After this early period, growth becomes slow in the hypopituitary dwarf and may continue for several decades, the epiphyses remaining open and the ultimate height being normal or shorter than normal.[25]

It is a well known fact that the individual stops growing after epiphyseal closure and that the presence of an eosinophilic adenoma in the sella tursica frequently results in gigantism before closure of the epiphyses and acromegaly after closure of the epiphyses; the epiphyses being closed, the long bones cannot lengthen further but become wider under the influence of the increased amounts of growth hormone. Evidence indicates that the normal adult produces growth hormone. Gershberg[6] has shown that the growth hormone content of the pituitary gland does not vary with age, and Daughaday[26] has shown that the serum levels of sulfation factor, a growth hormone dependent factor that stimulates in vitro uptake of S^{35} into cartilage of hypophysectomized rats, are similar in growing children and adults. Read,[15] by using a highly sensitive immunologic technique for measuring growth hormone in serum, has also found no difference in children and adults. Why does the normal adult, then, not have acromegalic features? One can explain this by stating that the sensitiveness of the adult tissues to growth hormone

is decreased, or that the anabolic action of growth hormone is counter-balanced by normal catabolic processes.

For years it has been accepted theory that the physiologic abnormality in acromegaly is increased production of growth hormone by the eosinophilic granuloma in the sella tursica. This theory was based on the findings in the disease which are similar to those that occur in animals treated with excessive amounts of growth hormone; these are skeletal enlargement, organomegaly, a tendency to hyperglycemia and glucosuria, and elevated serum phosphorus levels. Recent evidence strongly suggests that this theory may indeed be fact. Gemzell[27] has described a method for the concentration of a growth hormone-like factor in human plasma; a high concentration of this factor was found in patients with active acromegaly and no activity was found in normal controls and in cases of inactive acromegaly. Daughaday[26] has also found that the serum sulfation factor is increased in active acromegaly. Read[15] has also found elevated levels of growth hormone in acromegaly by use of his immunologic technique for measuring growth hormone.

On the other hand, clinicians have postulated that a deficiency in the production of growth hormone contributes to some of the findings in the various pituitary deficiency syndromes. These findings are failure of growth in children, insulin sensitivity and a tendency to hypoglycemia, and decrease in nitrogen storage. The sulfation factor described by Daughaday is low in patients with hypopituitary dwarfism, Sheehan's syndrome, and in patients with hypophysectomy.[26] Read[15] has found no evidence of growth hormone in three of four patients with hypophysectomy. Both of these methods of testing are very sensitive; the test described by Gemzell is not sensitive enough to show the low levels in hypopituitarism and normal growth.

In 1949, Cotes, Reid, and Young[36] and Houssay[37] showed the diabetogenic action of purified animal growth hormone in animals. Evidence suggests that human growth hormone is also diabetogenic.[30,31] Many have wondered if an excessive production of growth hormone could be causally related to diabetes mellitus. Diabetes mellitus is commonly associated with acromegaly and it seems not illogical to suppose that the hypersecretion of growth hormone could cause both syndromes. This could be one explanation for the large size of infants of diabetic mothers. These infants show gigantism, visceromegaly, pancreatic beta cell hyperplasia, and a tendency to have lower blood glucose levels than normal infants. One could explain the diabetes mellitus in the mother and gigantism in the infant as being due to increased production of maternal growth hormone, the amount being too low to cause acromegaly but high enough to cause diabetes and at the same time cause the infant to be large.[32] Forsman and Gemzell,[33] by using their method of concentrating growth hormone activity in human serum, have found elevated levels in twenty-four of forty patients with diabetes mellitus. The frequency of high levels was greater in juvenile diabetes and in young adults with diabetes. This finding adds new evidence to the theory that growth hormone is causally related to diabetes mellitus.

Some Clinical Aspects of the Use of Growth Hormone

Because human growth hormone is produced from the pituitary glands of autopsy material and thus scarce in supply, its usefulness as a therapeutic agent is limited to clinical research. Monkey growth hormone which is also physiologically active in man is limited in amount. One of the great disappointments in this field was the finding that animal growth hormone which is available in large enough amounts to be commercially available does not act in man. One great hope in this field is that animal growth hormone can be modified in such a way as to be active in man. Because of the complex nature of the molecule, it is unlikely that the hormone can be synthesized in the near future.

That human growth hormone would be useful in replacement therapy in pituitary deficiency is predictable, and has been shown to be true; two milligrams three times weekly produces a growth rate of 2.6 inches per year in pituitary dwarfism.[34] As an anabolic agent in premature infants, growth hormone

was shown to be ineffective.[35] Human growth hormone has failed to produce growth in chrondrodystrophia.[36] Crigler et al. found that 2 milligrams per day did not affect the blood sugars in a patient with hypoglycemia and hyperinsulinism even though there was an increase in serum non-esterified fatty acids and in positive nitrogen balance.[29] No toxic reactions have been reported.

There are two main problems in the field of growth hormone clinical research: The first of these is the development of a clinically feasible method of measuring growth hormone in human biologic fluids. Such a test would be extremely useful in the diagnostic work up of the many growth problems in children. It would be valuable in the diagnosis and management of hypopituitarism and acromegaly. This test might be helpful in working out the pathological mechanism involved in the etiology of diabetes and in explaining growth failure in such conditions as cardiac disease and renal disease in children. One frequently asked question concerning these diseases is: Is growth failure due to decreased production in growth hormone or it is due to a failure of diseased tissues to respond to the hormone? Some of the methods of measuring growth hormone activity in man were mentioned above; these are the immunologic method of Read,[13,15] the serum sulfation factor of Daughaday,[26] and the serum concentration method of Gemzell.[27,33] It is hoped that one of these may be refined sufficiently to be of clinical use.

The second great problem in growth hormone research is the finding of a product that is active in man and capable of being produced in sufficient quantity as to be commercially available. One promising aspect of this is the finding by Li that beef growth hormone, when partially digested by chymotrypin, will cause nitrogen retention in man.[10]

A growth hormone product that is physiologically active in man has many interesting therapeutic possibilities. Its linear growth producing property may make it useful in treating some of the many small children who seem to have no demonstrable evidence of pituitary insufficiency as well as those who have demonstrable pituitary insufficiency. Its protein anabolic effect may make it of value in the recovery stages of such nitrogen wasting states as burns, tuberculosis, and anorexia nervosa. It may prove useful in the management of uremia of renal failure by its power to prevent protein catabolism. It is possible that its skeletal effects may make it advantageous in the treatment of non-healing fractures.

Summary and Conclusion

Human growth hormone is a protein that can be extracted in small amounts from the human pituitary gland. Methods of estimating growth hormone activity in human serum are available. Its main physiologic action is protein anabolism. It causes nitrogen retention and contributes to linear growth in children. When produced in excessive amounts it causes gigantism before epiphyseal closure and acromegaly in adults. It may be causally related to diabetes mellitus through its diabetogenic action. Production is decreased in certain pituitary deficiency syndromes. Clinical trials to date indicate that it is useful as replacement therapy in pituitary dwarfism.

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(Continued on Page 211)

A case of lead intoxication that emphasizes an age old clinical entity, which is seldom stressed, yet continues to be a health problem.

LEAD INTOXICATION

E. EUGENE SMITH, M.D.*
EDWARD T. SHIRLEY, M.D.*

THE RECOGNIZED health hazards of lead containing paints has prompted a decrease in their usage, diminishing one source of lead intoxication. Because lead poisoning is not a reportable disease, its present incidence in Oklahoma cannot be accurately compared with that of twenty years ago. Although there has been a decrease in the incidence of the disease,[1] it has not been completely eradicated.

This decreasing incidence has frequently lulled physicians into omitting this condition from their differential diagnosis although the signs and symptoms are well known. Due in part to this, the general population is not aware of the hazards, sources, or many times, even the existence of lead intoxication.

The following is a case report of an individual who worked with lead containing materials, but was unaware of the potential danger.

Case Report

The patient, a twenty-six year old white male construction worker, noticed the insidious development of anorexia and weight loss late in 1958; twenty-two pounds by the time of hospitalization April 6, 1959. During January of 1959, he had the sudden onset of generalized myalgia with cramping muscular pains. This was shortly followed by insomnia, malaise, progressive weakness, more marked in the extremities, and a period of diarrhea with subsequent marked constipation. During the following week he experienced chills and fevers lasting three to four days which he attributed to influenza. It was during this time that he began to have occasional bouts with nausea and a metallic taste in his mouth. After approximately one month, his symptoms diminished with a return of near well-being.

Two weeks prior to hospital admission, in the latter part of March of 1959, increasing irritability and a recurrence of anorexia were noted. Within one week all the previous symptoms, except the chills and fever, had returned. From then until hospitalization there was a rapid progression of muscular weakness and cramping in the extremities.

The occupational history was pertinent in that this patient was a foreman on a construction team, and had, as one of his principal duties, the using of a spray gun to apply a fieldcoat paint, containing a lead base, to bridge beams. He had used this paint without benefit of a shield or mask for a period of two to three weeks in March, 1957, eight weeks in November and December, 1958, and the last time starting March 15, 1959 until the time of hospitalization on April 6, 1959. The past and family history were non-contributory. The review of systems was negative except for a history of allergy to penicillin.

Physical Examination: Blood pressure 120/70 mm/hg in the left arm supine, pulse 80 per minute and regular, respirations 16 per minute and temperature 99°F. The examination revealed a well developed, well

*E. Eugene Smith, M.D., a graduate of the University of of Oklahoma School of Medicine in 1959, was serving a third year preceptorship with Edward T. Shirley, M.D., in Wynnewood, Oklahoma when data was gathered for this paper. He is now a resident at the University of Oklahoma Hospitals.

nourished white male who appeared chronically ill. There was evidence of recent loss of muscle substance and a slight pallor. A lead colored line was present along the gum margins of both the upper and lower teeth and there was evidence of some pyorrhea. Generalized muscular tenderness and weakness were present, more marked in the muscle groups supplied by the radial and peroneal nerves. There was spasm of these muscle groups noted on exertion. On neurologic examination, the patient was found to be alert, orientated and cooperative. Cranial nerve functions were intact. Perception of pain and touch were within normal limits. Vibratory sense was diminished bilaterally in the extremities. Deep tendon reflexes were hypoactive except for the achilles reflexes, which were normally active. Hoffman and Babinski responses were absent.

Laboratory Findings on Admission: Hemoglobin was 13.7 gms.%, hematocrit 42%, W.B.C. 9,300 per mm³ with 2% eosinophils, 41% segmented neutrophils, 48% lymphocytes, and 9% monocytes. Four percent of the erythrocytes revealed basophilic stippling. The lead concentration in the urine on admission was found to be 0.53 mgm. per liter, with the upper limits of normal being 0.2 mgm. per liter.*

Hospital Course: He was considered to have chronic lead intoxication and was treated over a twelve day period with a total of 16 gms. of edathamil calcium-disodium, given intravenously in 5% glucose in water. On the second day of this treatment the urinary lead concentration was 6.25 mgm. liter. By the end of the first week of treatment his vibratory sense and deep tendon reflexes had returned to normal, his appetite had improved and his insomnia had disappeared. In addition, all evidence of muscle tenderness and spasm had disappeared.

On the fourteenth hospital day, the hemoglobin was 13 gm.% and again, 4% of the erythrocytes revealed basophilic stippling. At the time of discharge the neurologic examination was within normal limits, his strength was near normal, and he was having daily bowel movements.

*Urinary lead determinations done by the dithizonate extraction method (high pH extraction with bismuth.)

Discussion

Hippocrates, approximately 270 years B.C., was probably the first to recognize lead intoxication. It was not until the fifteenth century, however, that much interest was aroused in this clinical entity. This was brought about by the general use of metallic leads to correct the harsh acid taste in wine. In Germany such disastrous results followed the adulteration of wine that decrees were issued in the years 1498 and 1577 invoking the death penalty for adding lead to wine.[2] But it was not until the nineteenth century that the experimental studies of the action of lead in the body were pursued.

The disease as we know it today might be arranged in two categories; the childhood type and the adult variety. The manifestations of these clinical types are vastly different. The childhood type is characterized primarily by pica, and encephalopathy. It cannot be too strongly advised to consider plumbism in any child with a history of pica. Whether this is a cause and effect relationship or vice versa cannot be stated. If a high index of suspicion is not present early before the personality changes have become quite pronounced, it is only inviting disaster. Once convulsions have occurred, the disease most often progresses to death. In those cases where death does not ensue there is a high incidence of neurological sequela even in those instances where modern therapy has been employed. The most common source of intoxication in children today is from lead containing paints, notably flakes of wall plaster and paint from ceilings and woodwork.[3,10] There has been a marked decline in the number of reported cases of intoxication from painted toys and cribs. This has been mainly due to the education of manufacturers to the danger of lead containing paints and to legislation requiring the labeling of paint, as to whether it is to be used for indoor or outdoor use, and the percentage of lead it contains. Milder forms of plumbism in children appear to occur less frequently than the severe form, but this may be more apparent than real.

In adults, the symptomatology may be varied but is usually that of colicky abdominal pain or muscle cramps in the extremities, frequently affecting those muscle

groups most often used. This is accompanied by some degree of anorexia and constipation. Encephalopathy is a far less frequent component of the disease, than in childhood, even with chronic long standing exposure; the only exception being the acute form following tetraethyl lead intoxication. Encephalopathy in those rare cases where it does occur, is milder, less frequently accompanied by convulsion, and on subsiding usually leaves no residual neurologic deficit.[4, 6, 8, 11]

Of the various routes of entrance of lead into the body i.e., gastrointestinal, lungs and unbroken skin, the two most frequent are the gastrointestinal and the respiratory route.[5] The former being the chief means of lead intoxication in childhood and the latter in adults.

The severity and rapidity of onset of symptoms often depends on the route of entrance.[6, 7, 9] Early onset and more severe symptomatology are more frequently associated with the respiratory route. This may be explained in part by the ease of solubility of lead and lead oxide in blood plasma. When dust laden with these materials is breathed into the alveoli it has easy access to the plasma.[6, 7, 9] Another factor is that both the absorption and excretion of lead entering the body by way of the respiratory tract involves transportation in the systemic blood, while lead entering through the gastrointestinal tract may be excreted without being absorbed or after absorption may be trapped by the liver and excreted in the bile without ever reaching the systemic circulation.[6, 7, 9]

Thus it is apparent in our present society that lead intoxication is still a problem. The patient above presents one phase of this problem, intoxication via the respiratory route, which is generally considered to produce a more fulminant disease than absorption via the gastrointestinal route.

Summary

This has been a case presentation of a young white male who developed lead intoxication via the respiratory route following exposure to a lead containing paint. It is of special interest that although he was of the middle socio-economic class he was unaware of the potential danger of lead intoxication associated with his work.

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(Continued from Page 208)

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ABSTRACTS

Antimicrobial Agents

Summary of a Round Table Discussion

MARK H. LEPPER,* and HARRIS D. RILEY, Jr.**

Pediatrics, 23: 1192-1198, June, 1959

Department of Preventive Medicine, University of Illinois.
**Professor of Pediatrics.

Sleep Deprivation

JOHN H. BRAUCHI* and LOUIS J. WEST.**

J.A.M.A., 171: 11-14, Sept. 1959

The effects of prolonged wakefulness were studied in a man who had been hospitalized for nervous disorders on three previous occasions. After the third hospitalization he had worked successfully under exacting conditions for six years, until he subjected himself to two periods of sleep deprivation. The first period of 89 hours precipitated an acute state of confusion from which he quickly recovered. The second period produced hallucinations, delusions, and complete disorganization. It was terminated at 168 hours and 33 minutes, but the psychotic symptoms and personality changes persisted. His affairs became confused, and he sought treatment. After four months of hospitalization he returned to work. He has been in reasonably good health for more than a year since that time.

*Resident in Psychiatry, University of Kansas Medical Center.
**Professor and Head, Department of Psychiatry, Neurology, and Behavioral Sciences, University of Oklahoma School of Medicine.

Psychophysiology of Hypnosis

LOUIS J. WEST.*

J.A.M.A., 172: 672-674, Feb., 1960

Psychophysiologically, the hypnotic trance may be defined as a controlled dissociated state. This state of altered awareness is maintained through "Parassociative" mechanisms mediated by the ascending reticular activating system. These mechanisms probably involve the thalamocortical projection system, corticofugal systems both excitatory and inhibitory, and other integrated regulatory systems known to function in connection with the ascending reticular activating system. When the subject is in the hypnotic state, alertness is maintained relative to the inhibition or exclusion from awareness of considerable amounts of incoming information that would ordinarily be consciously perceived in the process of reality-testing. Under these circumstances the information inserted into the restricted area of the subject's awareness by the hypnotist, through his suggestions, is accepted as reality to a greater or lesser extent, depending on the subject's dissociation of other information from awareness, moment by moment. Recently observed bioelectric variations during the hypnotic state, as well as differing reactions by hypnotic subjects to drugs under various circumstances, are compatible with this theory, which is formulated primarily to stimulate further discussion and research.

*Professor and Head, Department of Psychiatry, Neurology, and Behavioral Sciences.

Epidemiologic Studies of Histoplasmin Sensitivity

II. Conversion Rates

HARRIS D. RILEY, Jr.*

American Practitioner and Digest of Treatment, 10: 1931-1939, Nov., 1959

Twenty-nine (12.4%) of 243 infants and children from all parts of the world who were initially histoplasmin negative on arrival at an Air Force base in middle Tennessee converted to positive on retesting. The rate of conversion increased with age but there was no seasonal influence. The conversion rate of individuals who had previously resided in areas of low incidence of histoplasmosis was similar to the group who were natives of endemic areas but too few in the former group were available for retesting to draw a valid conclusion. There was a greater tendency for conversions to occur when retests were performed at less than six months after the initial test than at longer intervals. This finding can be regarded as only tentative until larger groups residing in the same circumstances and locale are retested. The nature of histoplasmin sensitivity and other aspects of histoplasmin conversion rates are discussed. The findings relative to conversion rates are compared to those in other studies in other geographic areas.

*Professor of Pediatrics.

Primary Chickenpox (Varicella) Pneumonia: Report of Two Cases Seen in the Private Practice of Internal Medicine

JAMES J. GABLE, Jr.*

Ann. Int. Med. 51: 583-590, Sept. 1959

Primary chickenpox pneumonia is usually a disease of adults. It has been reported with increasing frequency in the last few years. Two additional cases encountered in the private practice of internal medicine are here reported. Patients with primary chickenpox pneumonia present a typical clinical and roentgenologic picture. Treatment consists of symptomatic management, with delivery of sufficient oxygen to the patient. ACTH and cortisone are contraindicated in patients with chickenpox or primary chickenpox pneumonia unless definite adrenal insufficiency exists. One can only speculate as to the significance of the apparent increase in primary chickenpox pneumonia during the past generation.

*Assistant Professor of Medicine.

FACULTY NEWS

New Appointments Named By Board of Regents

New members of the departments of radiology and psychiatry are among recent appointments to the full time faculty of the University of Oklahoma School of Medicine.


BRODIE

Hooshang Taybi, M.D., who recently completed a radiology residency at Kings County Hospital, Brooklyn, was named assistant professor of radiology and Charles M. Brodie, Ph.D., former teaching and research assistant at the University of Illinois, was appointed instructor in medical psychology.

University regents at the same time approved the appointment of Merlin K. DuVal, M.D., associate professor of surgery since 1957, as vice-chairman of that department.

Doctor Taybi, a 1944 graduate of Teheran University Medical School, interned at St. Vincent's Hospital, Staten Island, New York, took two years additional graduate work at Bellevue Medical Center and a residency in pediatrics at Children's Hospital, Akron, Ohio. He also served two years as a fellow in pediatric roentgenology at Cincinnati Children's Hospital after receiving a master of medical science degree in pediatrics at New York University in 1954.

Doctor Brodie is the first addition to the Psychiatry Department's division of medical psychology, headed by Doctor Oscar A. Parsons, who joined the faculty last fall. He was awarded his Ph.D. early this year at the University of Illinois. A graduate of Carleton College, he held Ford Foundation and United States Public Health Service fellowships while working toward his doctorate. His research includes collaboration with Doctor Floyd S. Cornelison, associate professor of psychiatry, on self-image experience studies. The staff of the department also includes a clinical assistant, Mrs. Nancy Adsett.

Other newcomers to the faculty are Johan Adolf Wulff, M.D., clinical assistant in medicine, and William J. Craig, M.D., clinical assistant in pediatrics. Doctor Wulff came here from Seattle, where he took a residency in internal medicine at Virginia Mason Hospital. Doctor Craig, a 1951 OU Medical School graduate, finished his residency at Children's Memorial Hospital January 1. Earlier he served two years in the U. S. Air Force and was a medical consultant to the State Department of Public Welfare three years.

G. Rainey Williams, M.D., Named Markle Scholar

G. Rainey Williams, M.D., assistant professor of surgery at the University of Oklahoma School of Medicine, is one of 25 young medical teachers in the United States and Canada recently appointed Markle Scholars in Medical Science for the five-year period beginning July 1.

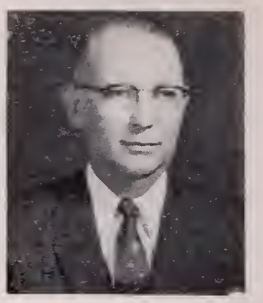

WILLIAMS

Each appointment carries with it a grant of $6,000 a year to supplement the Scholar's support and aid his research. The program was established by the John and Mary R. Markle Foundation of New York to strengthen medical education by offering promising young scientists academic security and financial help early in their careers.

Doctor Williams becomes the fourth Markle Scholar on the OU faculty. Others are: Gilbert S. Campbell, M.D., Ph.D., professor, Merlin K. DuVal, M.D., associate professor, and Doctor Rene Menguy, M.D., Ph.D., assistant professor, all of the Department of Surgery.

Doctor Williams received his M.D. at Northwestern University in 1951, interned and took his surgical residency at Johns Hopkins, and joined the OU faculty in 1958. He is a member of the team which developed the Medical Center's open heart surgery program and his major research interest also is cardiovascular surgery.

PRESIDENT'S LETTER



Show the physician due honor in view of your need of him,
For the Lord has created him;
Healing comes from the Most High,
And he will receive presents from the king.
The skill of the physician exalts him,
And he is admired among the great.
The Lord has created medicines out of the earth,
And a sensible man will not refuse them.
Was not water made sweet by wood,
So that its strength might be shown?
And he has given men knowledge
So that he might be glorified for his wonderful works.
With them he cures and takes away pain,
The druggist makes a mixture of them.
His works will never end,
And from him peace spreads over the face of the earth.

The Wisdom of Sirach—38: 5-8

Often in the past few years when I have been unable to comprehend and accept, and consequently become argumentative, relative to such things as rock and roll, progressive education, modern jazz dance, H. R. 4700, federal aid to education, increasing centralization of our government, and the increasing efforts to make everyone financially dependent upon the Federal Government, and the innumerable socio-economic factors influencing the practice of medicine, such as Medicare and the Old Age Medical Assistance Programs, I have been told repeatedly that I was born 50 years too soon.

Reading the above, taken from the Apocrypha, and written about 180 B.C., showing such a great appreciation of the physician, makes me feel that I was not born soon enough.

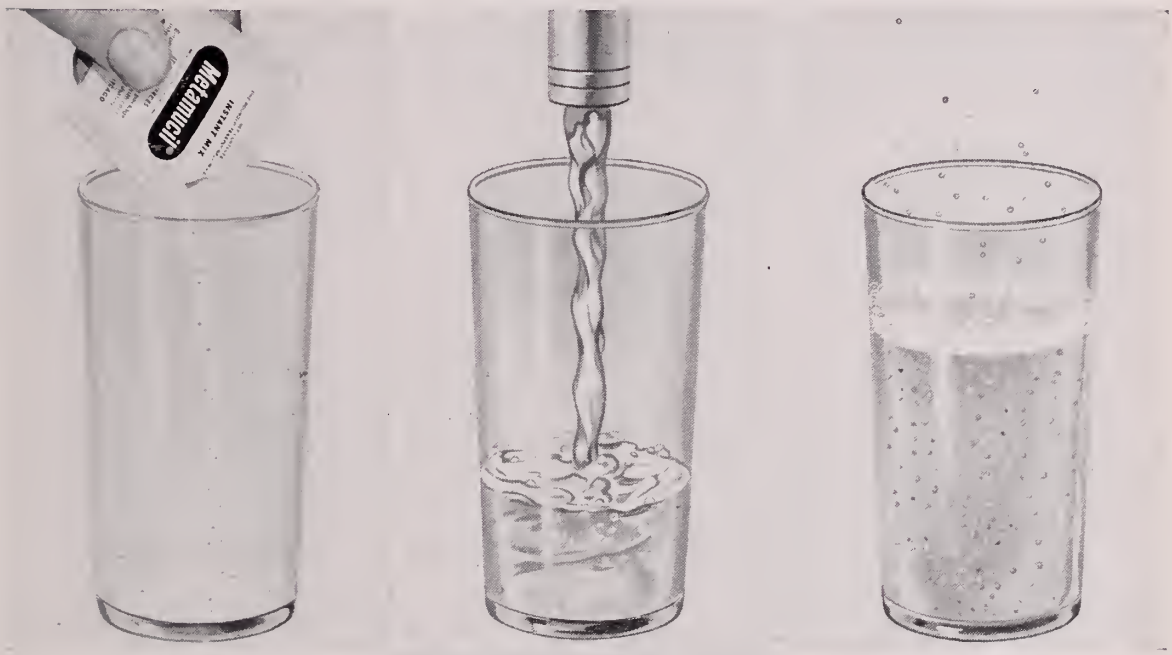
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Special Article

Factors Relating To Physician Distribution in Oklahoma 1950-1959

Part III of a Series**

ROBERT C. LOWE, M.D.*
JAMES A. HAGANS, M.D.*
PEARL FISHER, Ph.D.*

AN OUTSTANDING contemporary economist*** argues that education is not a social service but an industrial investment, upon which a measurable monetary profit can be realized in several ways. The same logic of our free enterprise profit system is being applied to academic, preventive, therapeutic and rehabilitative medicine by medical, governmental and industrial organizations. However, the individual community attitude, like that of the individual person, is still much like that of a consumer rather than that of an investor.

Urbanization is a qualitative development, depending upon attitudes as well as upon the degree of population concentration and organization. A satisfactory answer to the question previously stated in Part II, "Why does community size appear to be significant in the 'urbanized' counties but not in the others?" would require information on individual community efforts to improve their schools, recreation facilities, paving, sewage and garbage disposal and—particularly pertinent to this subject—their attitudes and actions in the individual or joint community management of their health affairs for the community and for the individual citizen. Traditionally, the latter has been largely left to the medical profession or agencies far removed from the scene. Even the providing of necessary hospital facilities, particularly in the middle west, has been left to physicians by many communities. There are presently 29 of the 77 counties in Oklahoma without any local health department services.

Almost 35% of the presently existing hospitals have been built or developed and operated by physicians needing such facilities to practice in a community of their choice.

However, the data in table VI, which demonstrates the influence of aging on the distribution of practicing physicians, will

*All three of the authors are with the Department of Preventive Medicine and Public Health, University of Oklahoma School of Medicine; Doctors Hagans and Fisher are also with the Biostatistical Unit of the Department of P.M. and P.H.; in addition, Doctors Lowe and Hagans serve with the Department of Medicine.

**Part II appeared in the March 1960 issue of this Journal.

***J. K. Galbraith—Men and Capital: Adventures of the Mind Series—Saturday Evening Post—March 5, 1960.

Table VI

INFLUENCE OF MIGRATION AND AGING ON PRACTICING M.D.'s, 1950-1959
In Relation to County Organization Levels and Community Size

Level of Organization	No. Counties	No. Towns	Col. 1 % M.D.'s Remaining	Col. 2 % M.D.'s New	Col.3 % Total Change	Col. 4 1950 % M.D.'s <60	Col. 5 1959 % M.D.'s <60	Col. 6 % Change <60	Col. 7 % Change >60 +
Metropolitan	2		69.8	37.1	+9.9	76.9	80.1	+15.5	—4.3
"Urbanized"	21		57.9	38.8	—5.9	63.1	72.9	+9.3	—30.4
<2500		51	26.0	48.9	—35.6	43.8	61.7	—9.4	—56.1
2500 - 6000		15	45.1	43.4	—25.4	57.7	66.0	—14.6	—40.0
6000 - 18000		13	60.2	35.0	—8.2	66.3	73.3	+1.5	—27.3
18000 - 38000		9	68.0	38.7	+9.8	67.4	75.5	+23.2	—17.7
Rural—I	9		58.0	28.4	—16.8	53.5	61.1	—7.5	—32.2
<2500		38	50.0	27.9	—30.6	33.9	27.9	—42.9	—24.4
2500 - 6000		4	56.0	36.4	—12.0	64.0	77.3	+6.3	—44.4
6000 - 18000		6	62.8	26.8	14.1	62.0	72.2	0.0	—37.2
Rural—II	45		46.9	37.0	—25.6	51.5	63.4	—8.5	—43.8
<2500		107	39.3	40.5	—33.0	40.5	47.4	—21.4	—40.8
2500 - 6000		25	54.3	34.8	—17.7	60.4	74.1	+1.0	—46.2
6000 - 18000		3	50.0	31.8	26.7	66.7	81.8	—10.0	—60.0

also throw some light on the significance or lack of significance of community size at the various organizational levels. In this table the physician population has been sorted according to (1) age below 60 years and (2) age 60 years and over.

Table VI—(Col. 6 and 7) demonstrates a net gain of M.D.'s in the younger age group for both the metropolitan and "urbanized" counties. On the other hand, a net loss, similar for both rural organization levels was observed. A net loss of physicians in the older age group was noted in all organizational levels. The loss is similar in degree for the "urbanized" and Rural—I levels. It is least in the metropolitan and greatest in the Rural—II levels.

A further break down of this same data for each organizational level and relative to community size (Col. 6 and 7) is given along with the per cent of physicians remaining in 1959 from 1950 (Col. 1) the per cent of physicians new since 1950 and remaining in 1959 (Col. 2), and the per cent of physicians less than 60 years of age in 1950 (Col. 4) and in 1959 (Col. 5). The figures for the total per cent change (Col. 3) are republished from Part II because they represent the net result of the combined changes of both age groups (Col. 6 and 7) and the net result of the combination of physicians remaining and new (Col. 1 and 2).

An optimistic note is struck by the fact that in all community sizes at all organization levels (Col. 4 and 5), (save Rural—I where there is essentially no change in the <2500 group) there has been an increase from 1950 to 1959 in the proportion of physicians under 60 years of age. This optimism may be questioned, however, when it is noted (Col. 6 and 7) that in only two areas (metropolitan and the larger "urbanized" communities of 18,000 to 38,000) was this the result of the net gains in physicians under age 60 overbalancing the net losses in physicians 60 years of age and over. In all other areas there was virtually no net increase in physicians under age 60, and in five there was actually a net loss. In all of these areas the increase in proportion of physicians under age 60 seen from 1950 to 1959 was simply the result of losing a smaller proportion of physicians from the population under age 60 than over that age. The infusion of younger blood into Oklahoma's practicing physician population is clearly

taking place primarily in the metropolitan and larger urbanized areas.

The significant relationship between the total net change of physicians and community size at the "urbanized" level of organization, as previously reported in Part II, can now be seen to result from the combination of a progressively decreasing net loss of physicians in the younger age group (which actually becomes a net increase in the larger communities) with a progressively decreasing net loss of physicians in the older age group in relation to increasing community size. It can also be noted, by comparison, that these same relationships to community size do not hold for either of the rural organization levels. A teleological explanation might be seen as rooted in the developmental history of these communities, some of which became urbanized in the sense outlined at the beginning of this paper, others of which remained rural. Further extensive research would be required to identify the pertinent variables in order to validate such an explanation.

The net decrease from the 1881 practicing medical physicians in the state of Oklahoma in 1950 to the 1795 in 1959 (-4.5%) represents the combined result of an 8.4% increase of M.D.'s less than 60 years of age and a decrease of 28.2% in those sixty years of age and over. This, of course, brings up the matter of supply.

From 1940 through 1955 (this cut-off date was chosen to allow for internship, additional training and/or military service) the University of Oklahoma School of Medicine graduated 1106 students of medicine. As of January 1, 1959, 497 (44.7%) were in active practice in Oklahoma. These results compare very favorably with those reported (2) from an extensive study in Illinois, a state with much greater resources. These Oklahoma students plus immigration of physicians from other states and schools resulted in a cumulative deficit of 86 practicing M.D.'s in nine years (1950 to 1959).

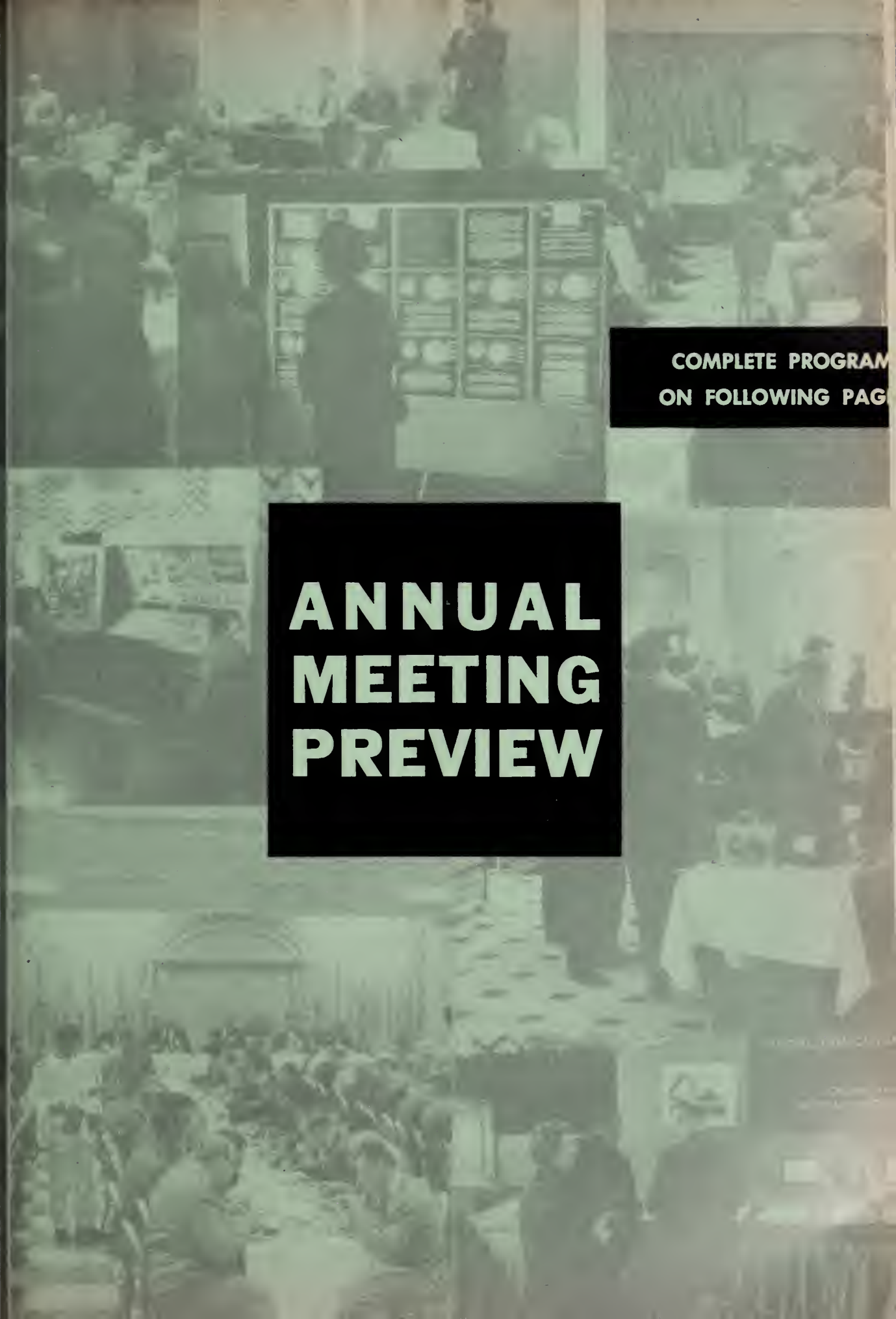
The core of the problem is not primarily the small deficit in practicing physicians noted above. Rather, the real problem is in attempting to evaluate whether the existing and similar future distribution of physicians in the state can meet a growing and broadening demand for medical care, in the face of the fact that no satisfactory measuring device exists, with which one can determine truly the quantitative adequacy of medical care as the division of labor has developed within the medical profession. Added emphasis is given to the significance of the changing distribution of physicians by the changing distribution of hospital beds which has taken place through new construction and additions since 1946. The distribution of new and existing hospitals and additional beds should imply a parallel relationship to the distribution of physicians to care for the number of potential patients upon which the operation and use of this bed capacity is predicated. For, indeed, of what use are hospitals without doctors? Part IV will attempt to show what relationship exists between these two distributions in Oklahoma.

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Councilor (1960)—J. W. Murphree, M.D., Ponca City

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Councilor (1961)—C. M. Hodgson, M.D., Kingfisher
Councilor (1961)—George T. Ross, M.D., Enid

District 4: Alfalfa, Beaver, Cimarron, Ellis, Harper, Major, Texas, Woods, Woodward
Councilor (1962)—Walter H. Dersch, M.D., Shattuck
Councilor (1962)—John X. Blender, M.D., Cherokee

District 5: Beckham, Blaine, Canadian, Custer, Dewey, Roger Mills
Councilor (1960)—Ross Deputy, M.D., Clinton
Councilor (1960)—C. Riley Strong, M.D., El Reno

District 6: Oklahoma
Councilor (1961)—Peter Russo, M.D., Oklahoma City
Councilor (1961)—E. E. Shircliff, M.D., Oklahoma City

District 7: Cleveland, Creek, Lincoln, Okfuskee, Pottawatomie, Seminole
Councilor (1962)—C. C. Young, M.D., Shawnee
Councilor (1962)—E. K. Norfleet, M.D., Bristow

District 8: Tulsa
Councilor (1960)—Wendell L. Smith, M.D., Tulsa
Councilor (1960)—Marshall O. Hart, M.D., Tulsa

District 9: Adair, Cherokee, McIntosh, Muskogee, Okmulgee, Sequoyah, Wagoner
Councilor (1961)—R. L. Currie, M.D., Stilwell
Councilor (1961)—Burdge F. Green, M.D., Stilwell

District 10: Haskell, Hughes, Latimer, LeFlore, Pittsburg
Councilor (1962)—Paul Kernek, M.D., Holdenville
Councilor (1962)—C. E. Lively, M.D., McAlester

District 11: Atoka, Bryan, Choctaw, Coal, McCurtain, Pushmataha
Councilor (1960)—Thomas E. Rhea, M.D., Idabel
Councilor (1960)—W. A. Hyde, M.D., Durant

District 12: Carter, Garvin, Johnston, Love, Marshall, McClain, Murray, Pontotoc
Councilor (1961)—William T. Gill, M.D., Ada
Councilor (1961)—M. E. Robberson, M.D., Wynnewood

District 13: Caddo, Comanche, Cotton, Grady, Jefferson, Stephens
Councilor (1962)—John B. Miles, M.D., Anadarko
Councilor (1962)—W. R. Cheatwood, M.D., Duncan

District 14: Greer, Harmon, Jackson, Kiowa, Tillman, Washita
Councilor (1960)—J. B. Hollis, M.D., Mangum
Councilor (1960)—R. R. Hannas, M.D., Sentinel

ANNUAL MEETING COMMITTEES



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General Chairman

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Program Committee Chairman
Bert F. Keltz, M.D.,
Scientific Works Committee Chairman
H. Thompson Avey, M.D.
Irwin H. Brown, M.D.
James P. Dewar, M.D.
Leonard P. Eliel, M.D.
Mark A. Everett, M.D.
George H. Guthrey, M.D.
Edmond H. Kalmon, M.D.
John W. Records, M.D.
Roger Reid, M.D.
H. K. Sowell, M.D.
J. R. Stacy, M.D.
Jim M. Taylor, M.D.
Ethan Allen Walker, Jr., M.D.

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Richard A. Clay, M.D.
James B. Eskridge, III, M.D.
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H. A. Ruprecht, M.D.

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TECHNICAL EXHIBITS COMMITTEE

Ancel Earp, Jr., M.D., *Chairman*
M. T. Buxton, Jr., M.D.
Farris W. Coggins, M.D.

DIGEST OF EVENTS



Hotel Accommodations

There will be no headquarters hotel for the Annual Meeting. Physicians are asked to make their own reservations at the hotel or motel of their choice. Among the hotels recommended are the Skirvin, Skirvin Tower and Biltmore. Indicate that you are attending the Annual Meeting when requesting reservations.

Registration

Registration will open Monday, May 2, in the Zebra Room of the Municipal Auditorium at 8:00 a.m. Delegates may register, both for the House of Delegates and the general meeting, in advance on Sunday, May 1, in the Hall of Mirrors of the Municipal Auditorium, beginning at 9:00 a.m.

Council

The Council of the Oklahoma State Medical Association will meet on Saturday, April 30, at 2:00 p.m. in the Regency Room of the Skirvin Hotel.

House of Delegates

The House of Delegates will meet on Sunday, May 1, at 10:00 a.m. in the Hall of Mirrors, Mezzanine, Municipal Auditorium. The session will recess in the early afternoon and will reconvene at an hour to be announced by the Speaker of the House. During the recess, a buffet lunch will be served in the Hall of Mirrors, at $1.50 per person. The Hall of Mirrors may be conveniently reached by the elevators at the north end of the Auditorium lobby.

Scientific Sessions

General Scientific Sessions will be held in the Zebra Room of the Auditorium all day Monday and Tuesday, and until noon on Wednesday. A complete program for the scientific sessions appears in this issue of *The Journal.* AAGP credit: 15 hours, category II. In addition to the general sessions, a number of specialty societies will have meetings, as described on page 238.

Luncheons

A Roundtable Luncheon will be held on Monday, from 12:20 p.m. until 1:30 p.m. in the Hall of Mirrors, and Mr. Gerald J. Skibbins, public opinion expert, will speak at a general luncheon in the Hall of Mirrors on Tuesday, from 12:30 p.m.-2:00 p.m. Tickets for both luncheons should be purchased at the Registration Desk at the time of registering for the meeting.

Scientific Exhibits

A selection of informative scientific exhibits by individual physicians and medical and health organizations will be on display in the Zebra Room of the Auditorium from 8:30 a.m. to 3:30 p.m. on Monday; from 8:30 a.m. to 5:30 p.m. on Tuesday; and from 8:30 a.m. until noon on Wednesday. Convention visitors are urged to inspect these educational exhibits, a complete list of which appears in this issue of *The Journal*.

Technical Exhibits

Fifty-five displays by firms offering products and services of interest to Oklahoma physicians will be featured at the 54th Annual Meeting. These Technical Exhibits will be located in the Zebra Room of the Auditorium, and may be seen from 8:30 a.m. to 3:30 p.m. on Monday; from 8:30 a.m. to 5:30 p.m. on Tuesday; and from 8:30 a.m. until noon on Wednesday. A complete list of exhibitors appears elsewhere in this issue of *The Journal*. Physicians are urged to visit all of these displays which are prepared by firms who contribute much to the profession and make the Annual Meeting possible.

Physicians Hobby Show

The Annual Physicians Hobby Show, a delightful and impressive display of the crafts and hobbies of Oklahoma physicians, will be located in the Regency Room of the Skirvin Hotel and will be open all day Monday and Tuesday, and a half day on Wednesday.

Sponsored by the Woman's Auxiliary, the Hobby Show will also be open during the Social Hour which will be held in adjacent rooms on Tuesday evening.

Golf and Tennis Tournaments

The Annual Golf and Tennis Tournaments will be held on Wednesday, May 4, beginning at 1:00 p.m.

Golfing will take place at beautiful Twin Hills Country Club, 3401 N.E. 36th Street. Following the tournament, a social hour and dinner will be held at 7:00 p.m. in the Clubhouse. Golf Trophies will be presented in all classifications during the dinner. The greens fee is $3.00 and the dinner $3.50.

A tennis tournament will be played at the Oklahoma City Tennis Club, 5600 N. Portland Avenue.

Physicians should register for these events at the Annual Meeting Registration Desk, Municipal Auditorium.

Past-Presidents' Breakfast

The Annual Past-Presidents' Breakfast will be held at 8:00 a.m., Monday, May 2, at the Oklahoma Medical Research Foundation, 825 N.E. 13th Street. For those who may need transportation, a Foundation station wagon will be at the Drive-In Entrance of the Skirvin Hotel at 7:30 a.m.

Inaugural Dinner-Dance

Beginning at 6:00 p.m. on Tuesday, May 3, with a Social Hour and Reception in the Balinese and Crystal Rooms of the Skirvin Hotel, the President's Inaugural Dinner-Dance will feature an evening of entertainment as well as the traditional installation ceremonies. Paul Neighbors, "The Prince of Personality," and his orchestra will furnish the music for dancing.

The Dinner-Dance will be held in the Persian Room of the Skirvin Tower Hotel, beginning at 7:00 p.m. Dancing will follow the dinner and installation ceremonies, from 9:00 p.m. until 1:00 a.m.

Distinguished Guest Speakers

EDGAR V. ALLEN, M.D.
Rochester, Minnesota



Professor of Medicine, Mayo Foundation, Senior Consultant in Medicine, Mayo Foundation. Medical Degree, University of Nebraska, Class of 1925. Diplomate, American Board of Internal Medicine. Fellow, American College of Physicians. Member, Association of American Physicians, Central Society For Clinical Research (President, 1948), American Society for Clinical Investigation, Board of Directors, American Heart Association (President, 1956-57), American Foundation for High Blood Pressure Research, Section on Cardiovascular Diseases, National Research Council (1940-42, 1946-50). Awarded American Heart Association's Distinguished Service Medal, 1957, and Gold Heart Award, 1959. *Sponsor: Minard F. Jacobs, M.D., Oklahoma City.*

GRAYSON CARROLL, M.D.
St. Louis, Missouri



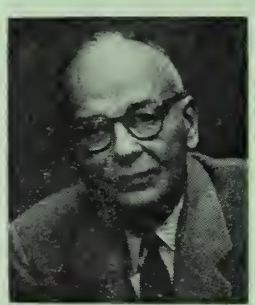
Professor of Urology, St. Louis University School of Medicine. Chief of Urology, St. John's Hospital, St. Louis. Medical Degree, University of Texas, Medical Branch, 1919. Diplomate and President, American Board of Urology. Member, American Association of Genito-Urinary Surgeons, American Urological Association (Treasurer), American College of Surgeons, International Urological Society, World Medical Association, New York Academy of Science, Southern Medical Association. Past Chairman, Urological Sections of American Medical Association and Southern Medical Association. *Sponsor: Jim M. Taylor, M.D., Oklahoma City.*

NADENE COYNE, M.D.
New York City, New York



Associate Professor, Physical Medicine and Rehabilitation, New York University—Bellevue Medical Center. Director of Education in Physical Medicine and Rehabilitation, N.Y.U.—Bellevue Medical Center. Director, Respirator Center, Goldwater Memorial Hospital. Medical Degree, University of Illinois Medical School, 1950. Previously certified as physical therapist and Instructor in Physical Therapy at Northwestern University. Diplomate, American Board of Physical Medicine and Rehabilitation. Member, American Congress of Physical Medicine and Rehabilitation, American Academy of Physical Medicine and Rehabilitation, American Physical Therapy Association, American Association of Electromyography and Electrodiagnosis. *Sponsor: Ella Mary George, M.D., Oklahoma City.*

GEORGE G. DEAVER, M.D.
New York City, New York



Professor of Clinical Rehabilitation and Physical Medicine, New York University—Bellevue Medical Center. Director, Children's Department of the Institute of Physical Medicine and Rehabilitation. Director, Department of Physical Medicine, Lenox Hill Hospital. Director, Bergen County New Jersey Cerebral Palsy Clinic. Consultant, St. Charles Hospital, New York, Hackensack Hospital, New Jersey and Crotched Mountain Children's Hospital, New Hampshire. Consultant to Surgeon General, United States Army. Medical Degree, University of Pennsylvania School of Medicine, 1917. Diplomate, American Board of Physical Medicine and Rehabilitation. Member, American Academy of Physical Medicine and Rehabilitation, National Society for Crippled Children and Adults, Professional Advisory Committee, N. Y. State Society for Crippled Children. Founding member and past-president, American Academy for Cerebral Palsy. *Sponsor: Herbert Kent, M.D., Oklahoma City.*

MICHAEL E. DeBAKEY, M.D.
Houston, Texas



Professor and Chairman of the Department of Surgery, Baylor University College of Medicine. Surgeon-in-Chief, Jefferson Davis Hospital, Houston, Senior Attending Surgeon, Methodist Hospital, Houston. Medical Degree, Tulane University, 1932 (M.S., 1935). Diplomate, American Board of Surgery, American Board of Thoracic Surgery. Member, American College of Surgeons, American Surgical Association, International Surgical Society, Society for Vascular Surgery (President, 1954), American Association for Thoracic Surgery (President, 1959), International Cardiovascular Society (President, 1959), Southwestern Surgical Congress (President, 1952), and others. Awards, Rudolph Matas Award in Vascular Surgery, 1954, AMA Hektoen Medal, 1954, Modern Medicine Award, 1957, I.S.S. Distinguished Service Award, 1958, Rosewell Park Medal, 1959, AMA Distinguished Service Award, 1959. *Sponsors: J. P. Wolff, M.D., Oklahoma City and Irwin H. Brown, M.D., Oklahoma City.*

EUGENE L. DERLACKI, M.D.
Chicago, Illinois



Associate Professor, Department of Otolaryngology, Northwestern University Medical School. Senior Attending Staff, Chicago Wesley Memorial Hospital. Medical Degree, Northwestern University Medical School, 1939. Diplomate, American Board of Otolaryngology. Member, American Academy of Ophthalmology and Otolaryngology (Member of Board of Secretaries and Program Committee, Secretary for Instruction Program and Scientific Exhibits), American Society of Ophthalmologic and Otolaryngologic Allergy, American College of Allergists, American Laryngological, Rhinological and Otological Society, American Otological Society, Pan-Pacific Surgical Association. *Sponsors: Dick M. Lowry, M.D., Oklahoma City, and Ethan Allen Walker, Jr., M.D., Oklahoma City.*

VINCENT J. DERBES, M.D.
New Orleans, Louisiana



Professor of Dermatology and Director of Division of Allergy and Dermatology, Tulane University School of Medicine. Medical Degree, Tulane University, 1934. Diplomate, American Board of Dermatology and American Board of Internal Medicine (Subspecialty of allergy). Member, American College of Allergists, American College of Physicians, American Academy of Allergy, Society of Experimental Biology and Medicine, American Academy of Dermatology and Syphilology, Society of Investigative Dermatology, Mediaeval Academy of America. Editorial Boards, Annals of Allergy and Quarterly Review of Allergy and Applied Immunology. *Sponsor: Mark A. Everett, M.D., Oklahoma City.*

MURDINA M. DESMOND, M.D.
Houston, Texas



Associate Professor, Department of Pediatrics, Baylor University College of Medicine. Head, Newborn and Premature Section, Jefferson Davis Hospital. Associate in Pediatrics, Methodist Hospital. Pediatric Consultant, Department of Obstetrics, Baylor, and Pediatrician, Junior League Well Baby Clinic, Hermann Hospital, Houston. Medical Degree, Temple University School of Medicine, Class of 1942. Diplomate, American Board of Pediatrics. Member, American Society for Pediatric Research, The American Society for Mental Deficiency. *Sponsor: Lucius Waites, Jr., M.D., Oklahoma City.*

BENJAMIN FELSON, M.D.
Cincinnati, Ohio



Professor and Director of Radiology, University of Cincinnati. Director, Departments of Radiology, Cincinnati General, Drake, Children's and Dunham Hospitals. Medical Degree, University of Cincinanti College of Medicine, 1935. Diplomate, American Board of Radiology, Member, Radiological Society of Greater Cincinnati, Ohio State Radiological Society, Radiological Society of North America (First Vice-President, 1959-60), American College of Radiology (Fellow), Association of University Radiologists, American College of Chest Physicians (Fellow). Honorary member, Texas, Philadelphia and Detroit Radiological Societies. *Sponsor: Samuel M. Glasser, M.D., Oklahoma City.*

KEN RAWLINSON
Norman, Oklahoma



Athletic Trainer, University of Oklahoma. Education, B.S. and M.S. Degrees from the University of Illinois in Physical Education. Past President of the National Athletic Trainers Association and the only trainer appointed to the NCAA's Committee on Sports Injuries and Safety. Selected as Trainer for United States' athletes at the 1960 Olympic Games, scheduled for Rome in August and September. He has conducted training clinics in fifteen states and two foreign countries and has authored many articles on athletic training, including those published in *The Journal* of the American Medical Association and *The Journal* of the Oklahoma State Medical Association. Recipient of "Trainer of the Year Award," Rockne Club of Kansas City, 1960. In 1959, he appeared on the program of the American Medical Association, Atlantic City. *Sponsor: Don H. O'Donoghue, M.D., Oklahoma City.*

SCHUYLER G. KOHL, M.D.
Brooklyn, New York



Associate Professor, Department of Obstetrics and Gynecology, Assistant Dean, State University of New York College of Medicine. Lecturer in Public Health and Administrative Medicine, Columbia University. Medical Degree, University of Maryland School of Medicine, 1940. M.S. and Dr. P.H. degrees, Biostatistics, Columbia University, 1952-1954. Diplomate, American Board of Obstetrics and Gynecology. Member, Brooklyn Gynecological Society, New York Academy of Medicine, American College of Obstetricians and Gynecologists Chairman, Committee on Maternal Welfare), Association of American Medical Colleges, Society for Gynecologic Investigation, New York Obstetrical Society, American Public Health Association. Consultant, Children's Bureau, AMA Committee on Maternal Welfare, National Institute of Neurological Diseases and Blindness. *Sponsor: Thomas C. Points, M.D., Oklahoma City.*

HAROLD ROSEN, M.D.
Baltimore, Maryland



Assistant Professor of Psychiatry, Johns Hopkins University School of Medicine. Chairman, Committee on Hypnosis, Council on Mental Health, American Medical Association. Education, B.A. in Greek, 1930, M.A. in Linguistics, 1931, Ph.D. in Indo-European Philology, 1933, University of Pennsylvania. Medical Degree, University of Pennsylvania School of Medicine, 1940. Diplomate, American Board of Psychiatry. Director, Maryland Psychiatric Society. Fellow, American Psychiatric Association and American Association for the Advancement of Science. Member, American Psychosomatic Society, Society for Clinical Experimental Psychiatry, International Society of Psychology, Maryland Association of Private Practicing Psychiatrists (Past President). Author of standard text, "Hypnotherapy in Clinical Psychiatry" and Editor of the book, "Therapeutic Abortion: Medical, Psychiatric, Legal, Religious and Anthropological Problems." *Sponsors: George H. Guthrey, M.D., Oklahoma City and L. J. West, M.D., Oklahoma City.*

GERALD J. SKIBBINS
Princeton, New Jersey



Research Executive, Opinion Research Corporation, Princeton, New Jersey. Liberal Arts and Journalism education, Northwestern University and the University of Illinois. Experience, industrial development consultant, trade association management, owner of direct sales merchandising company. Present duties, writer and lecturer on sales management, theory of government, economics and philosophy; specialty, major attitude research projects on medical public relations, political issues, marketing strategy. He is also especially interested in devising Executive Control systems for superior management problem-solving efforts of the future. *Sponsor: R. Q. Goodwin, M.D., Oklahoma City.*

MARIO STEFANINI, M.D.
Boston, Massachusetts



Associate Professor of Medicine, Tufts University Medical School. Director of Research and Hematologist, St. Elizabeth's Hospital, Boston. Medical Degree, University of Rome Faculty of Medicine, 1939. M.S., Biochemistry, Marquette University, 1947. Diplomate, American Board of Pathology. Member, American Society for Clinical Investigation and the American Society for Experimental Pathology. Formerly, Director of the Department of Clinical Laboratories, Boston Dispensary, and Physician and Hematologist, New England Center Hospital, Boston. He has contributed to 182 publications and has written two books. *Sponsor: Mark R. Johnson, M.D., Oklahoma City.*

HAROLD A. SOFIELD, M.D.
Oak Park, Illinois



Professor of Orthopaedic Surgery, Northwestern University, Chief Surgeon, Shriners Hospital for Crippled Children, Chicago, and the VA Hospital, Hines, Illinois. Consultant, Surgeon General, U.S. Army, and to Veterans Administration, Washington, D.C. Medical Degree, Northwestern University, 1928. Diplomate, American Board of Orthopaedic Surgery (Past President and Secretary). Member, American Orthopaedic Association, American College of Surgeons, American Academy of Orthopaedic Surgeons, Clinical Orthopaedic Society, International Society of Orthopaedic Surgery and Traumatology, Orthopaedic Research and Education Foundation (Secretary-Treasurer). Former Chairman of the Committee on Orthopaedic Surgery. National Research Council, and Special Consultant to the Surgeon General, Air Force. Associate Editor, Journal of Bone and Joint Surgery. *Sponsors: J. R. Stacy, M.D., Oklahoma City and Howard B. Shorbe, M.D., Oklahoma City.*

ANNUAL MEETING
TELEPHONE MESSAGE CENTER

While you are attending the Annual Meeting, your emergency calls may be referred to

CE 2-7206

Located at the Registration Desk,
Zebra Room Municipal Auditorium

Courtesy of

MEDICAL SERVICE SOCIETY
Of AMERICA

Monday, May 2, 1960

MORNING SESSION, Zebra Room, Municipal Auditorium
Alfred T. Baker, M.D., Durant, Presiding

9:00 a.m. SOME FUNDAMENTALS OF CARDIAC ROENTGENOLOGY
Benjamin Felson, M.D., *Cincinnati, Ohio*

9:30 a.m. THERAPY OF CONDUCTION DEAFNESS
Eugene L. Derlacki, M.D., *Chicago, Illinois*

10:00 a.m. PRESENT CONCEPTS OF PYELONEPHRITIS AND CHRONIC PAPILLARY RENAL NECROSIS
Grayson Carroll, M.D., *St. Louis, Missouri*

10:30 a.m. INTERMISSION—VISIT EXHIBITS

10:40 a.m. TYPES OF DISEASES SEEN IN THE NEWBORN
Murdina M. Desmond, M.D., *Houston, Texas*

11:10 a.m. PREVENTION OF ATHLETIC INJURIES, IMPROVED CARE OF THE HIGHSCHOOL ATHLETE
Ken Rawlinson, *Norman, Oklahoma*

11:40 a.m. HYPNOSIS IN MEDICAL PRACTICE
Harold Rosen, M.D., *Baltimore, Maryland*

12:20 p.m. ROUNDTABLE LUNCHEON—Hall of Mirrors
Alfred T. Baker, M.D., Durant, Presiding

Guest Participants: Benjamin Felson, M.D., Eugene L. Derlacki, M.D., Grayson Carroll, M.D., Murdina M. Desmond, M.D., Ken Rawlinson, Harold Rosen, M.D.

AFTERNOON SESSION, Zebra Room
Walter E. Brown, M.D., Tulsa, Presiding

1:30 p.m. THE ROLE OF ALLERGY IN OTOLARYNGOLOGY
Eugene L. Derlacki, M.D., *Chicago, Illinois*

2:00 p.m. UROLOGICAL CONDITIONS IN CHILDREN
Grayson Carroll, M.D., *St. Louis, Missouri*

2:30 p.m. THE ROENTGEN DIAGNOSIS OF PULMONARY COLLAPSE
Benjamin Felson, M.D., *Cincinnati, Ohio*

3:00 p.m. THERAPEUTIC ABORTIONS
Harold Rosen, M.D., *Baltimore, Maryland*

3:30 p.m.—Evening SPECIALTY SOCIETY MEETINGS
See Page 238

Tuesday, May 3, 1960

MORNING SESSION: Zebra Room, Municipal Auditorium
Bert F. Keltz, M.D., Oklahoma City, Presiding

9:00 a.m. THE NATURAL HISTORY OF ARTERIOSCLEROSIS OBLITERANS
Edgar V. Allen, M.D., *Rochester, Minnesota*

9:40 a.m. INTRAVASCULAR THROMBOSIS: HOW DOES IT OCCUR IN VIVO?
Mario Stefanini, M.D., *Boston, Massachusetts*

10:20 a.m. RECENT DEVELOPMENTS IN THE SURGICAL APPROACH TO PERIPHERAL VASCULAR DISEASE
Michael E. DeBakey, M.D., *Houston, Texas*

11:00 a.m. INTERMISSION

11:15 a.m. PANEL: "COAGULATION, CLAUDICATION AND CHIRURGERY"
Panelists: Edgar V. Allen, M.D., Mario Stefanini, M.D., Michael E. DeBakey, M.D.
Moderator: William Jaques, M.D., *Oklahoma City*

12:30 LUNCHEON—Hall of Mirrors
R. Q. Goodwin, M.D., Oklahoma City, Presiding

HOW PUBLIC OPINION CAN SOCIALIZE A PROFESSION OR INDUSTRY
Gerald J. Skibbins, Research Executive, Opinion Research Corporation, *Princeton, New Jersey*

AFTERNOON SESSION: ZEBRA ROOM
A. C. Lisle, M.D., Oklahoma City, Presiding

2:00 p.m. THE LIMPING CHILD
Harold A. Sofield, M.D., *Oak Park, Illinois*

2:30 p.m. RECENT ADVANCES IN THE TREATMENT OF FUNGUS DISEASES
Vincent J. Derbes, M.D., *New Orleans, Louisiana*

3:00 p.m. PERINATAL MORTALITY: PANEL DISCUSSION
Panelists: Schuyler Kohl, M.D., *Brooklyn, New York;* Murdina M. Desmond, M.D., *Houston, Texas;* Carl W. Lindstrom, M.D., *Tulsa;* Thomas C. Points, M.D., *Oklahoma City;* G. R. Russell, M.D., *Tulsa*
Moderator: Doctor Kohl

4:10 p.m. INTRAMEDULLARY FIXATION OF FRACTURES
Harold A. Sofield, M.D., *Oak Park, Illinois*

4:40 p.m. VESICOBULLOUS ERUPTIONS
Vincent J. Derbes, M.D., *New Orleans, Louisiana*

Wednesday, May 4, 1960

MORNING SESSION: Zebra Room, Municipal Auditorium
J. R. Stacy, M.D., Oklahoma City, Presiding

SPECIAL PROGRAM ON REHABILITATION

9:00 a.m. HEMIPLEGIA
Speaker: George G. Deaver, M.D., *New York City, New York*
Case Presentations and Patient Demonstrations: Ella Mary George, M.D., and Herbert Kent, M.D., *Oklahoma City*
Discussion: Doctors Deaver and Coyne

10:00 a.m. PARAPLEGIA
Speaker: Nadene Coyne, M.D., *New York City, New York*
Case Presentations and Patient Demonstrations: Shelby G. Gamble, M.D., *Okmulgee*
Discussion: Doctors Deaver and Coyne

11:00 a.m. PANEL ON TOTAL REHABILITATION
Panelists: Physical medicine and rehabilitation—Doctors Deaver and Coyne; Neurology—Richard E. Carpenter, M.D., *Oklahoma City;* Orthopaedic Surgery—J. R. Stacy, M.D., *Oklahoma City;* Psychology—Vernon Sisney, Ph.D., *Oklahoma City;* Urology—J. Hartwell Dunn, M.D., *Oklahoma City*
Moderator: Earl D. McBride, M.D., *Oklahoma City*

The Scientific . . .

ETHAN ALLEN WALKER, JR., M.D.
Oklahoma City

"Non-Radio Opaque Foreign Bodies of the Respiratory Tree in Small Children"

This exhibit will contain x-rays and foreign bodies, with a short history of the cases and diagnostic points.

VETERANS ADMINISTRATION HOSPITAL
Physical Medicine and Rehabilitation Service
OREN T. SKOUGE, M.D., Manager
ELLA MARY GEORGE, M.D., Chief, PM & R Service
Oklahoma City

"Rehabilitation of the Cerebrovascular Accident"

Photographs and paintings will be used to show that rehabilitation starts as soon as patient regains consciousness and continues until ready for discharge. Team working with patient includes physicians, nurses, physical therapist, occupational therapist, corrective therapist, speech pathologist, social service worker, psychologist and often employer and vocational rehabilitation counsellor.

WILLIAM R. RICHARDSON, M.D.
IRWIN H. BROWN, M.D.
Oklahoma City

"Neurogenic Megacolon — An increasingly Common Diagnosis in Oklahoma"

Personal case series of the authors will be shown with x-rays, photographs, charts and graphs. Emphasis will be placed upon the diagnosis and treatment in early infancy.

J. NEILL LYSAUGHT, M.D.
Oklahoma City

"Staphylococcal Infections in Children"

Exhibit will summarize experience with 59 cases of Staphylococcal infection in children and treatment with Furaltadone. Case descriptions, sensitivity studies and results will be depicted.

J. R. STACY, M.D.
Oklahoma City

"Bicipital Tenosynovitis"

This display will be comprised of anatomical drawings, photographs, charts and x-rays.

AVERILL STOWELL, M.D.
Tulsa

"Scalenus Anticus Syndrome and Neck Pain"

Anatomy and physiology of brachial plexus, cervical spine and neck muscles will be described with posters, x-rays and written descriptions.

JOSEPH W. KELSO, M.D., and JOSEPH W. FUNNELL, M.D.
Oklahoma City

"Gynecological - Urological Complications and Their Management"

This exhibit shows the management of all urinary injuries at the time of surgery, and delayed complications.

MARK A. EVERETT, M.D.
Oklahoma City

"Who Has VD?"

Epidemiology of an outbreak of syphilis and gonorrhea in a community. Presented with a diorama, charts and pictures.

ARTHRITIS AND RHEUMATISM FOUNDATION
Oklahoma City

"This Is Arthritis"

The exhibit illustrates the progression of rheumatoid arthritis from the normal to the fused joint. Professional literature and booklets for patients will be available for distribution.

ARTHUR H. DAVIS, M.D.
Tulsa

"Enucleation of Eyeball and Insertion of Troutman Magnetic Prothesis"

A color movie will be shown of the operation and insertion of the artificial eye.

. . . and Educational Exhibits

GEORGE L. WINN, M.D. and HERBERT KENT, M.D.
Oklahoma Allergy Clinic
Oklahoma City

"Pulmonary Function Tests in Asthma and Emphysema Patients"

Demonstration of the use of a 13.5 liter recording spirometer and electronic pneumotachograph. Graphs and tracings will be shown, as well as motion pictures pertaining to breathing exercises.

OKLAHOMA TUBERCULOSIS ASSOCIATION
Oklahoma City
OKLAHOMA TRUDEAU SOCIETY

"Vital Capacity: Its Measurement and Usefulness"

Graphs and charts will be shown on this subject and the vital capacity of those attending will be measured.

GLEN CAYLOR, M.D., GILBERT S. CAMPBELL, M.D.,
WILLIAM R. RICHARDSON, M.D. & G. RAINEY WILLIAMS, M.D.
University of Oklahoma Medical Center
Oklahoma City

"Management of Congenital Cardiovascular Anomalies in Infancy"

An illustration of problems, diagnostic techniques and surgical methods of treating cardiovascular disease in infancy. Physiologic recordings, photographs and x-rays will be used.

OKLAHOMA STATE DEPARTMENT OF HEALTH
Oklahoma City

"Multiphasic-Chronic Disease Screening"

A public screening program will be illustrated by actually performing tests on physicians and their wives. Screening procedures: Height and weight; 70 mm. chest x-ray; Clinitron blood sugar; Tuberculin test; Blood pressure; and, 6-lead electrocardiogram. Results will be available the next day after screening, complete with x-ray and ECG tracing for the physician to see. Screening results not picked up will be mailed to address indicated by physician.

OKLAHOMA POISON INFORMATION CENTER
Oklahoma City

"Organic Phosphorus Insecticide Poisoning"

Timely information will be furnished on the mechanism of organic phosphorus insecticide poisoning, symptoms produced, suggested treatment and mechanism of antidotal procedures.

AMERICAN CANCER SOCIETY
Oklahoma City

"Esophageal Speech Following Laryngectomy"

Various training courses and rehabilitation devices used with the laryngectomy patient are shown. An animated picture of the oral and nasal cavities, the pharynx, the esophagus and the upper portion of the stomach is used to show the varying depths to which an air bubble may be swallowed.

THE NATIONAL FOUNDATION
Oklahoma City

"The Changing Polio Picture"

This display presents data on the effectiveness of the present Salk vaccine and outlines a plan to eliminate unvaccinated "soft spots" in this country.

JACK WELSH, M.D., STEWART WOLF, M.D., and
LOIS RUSSELL, B.S.
University of Oklahoma Medical Center
Oklahoma City

"Fractionation of Large Molecular Constituents of Human Gastric Juice"

A presentation of fractionation patterns of human gastric juice in normal individuals and patients with a variety of disease states.

AMERICAN MEDICAL ASSOCIATION
Chicago, Illinois

"Foods in Oral Electrolyte Therapy"

This exhibit deals with the conditions that cause deviation in the electrolyte balance. The usefulness of foods in restoring the body's fluid balance is stressed.

COMMITTEE ON CLINICAL ANESTHESIA STUDY
Oklahoma Society of Anesthesiologists

"Curbstone Consultations on Anesthesia"

Staffed by members of the Society, the exhibit will present pictures of anesthetic procedures and complications and offer viewers an opportunity for conference. Printed materials will also be distributed.

W. T. McCOLLUM, M.D. and JOHN R. DANSTROM, M.D.
Mercy Hospital Heart Center
Oklahoma City

"Congenital and Acquired Cardiovascular Diseases"

The exhibit will present unusual and usual diagnostic problems relating to congenital and acquired heart disease, including electrocardiograms, x-rays, physiological studies, biplane angiocardiograms and cineradiography. Its purpose will be to project the general knowledge of the subject for use in clinical evaluation.

COMMITTEE ON THE ADVANCEMENT OF AUTOMOBILE SAFETY

American Academy of Orthopaedic Surgeons

"Prevention of Automobile Injuries"

The practical value of seat belts in the prevention of injury and death will be presented. Exhibit will also show effects of automobile posture upon somnolence and fatigue. Mechanism of neck injuries from rear end collisions and the testing of seat belts at the Sebring Sports Car Races will complete the display.

MARTIN M. CUMMINGS, M.D.
Department of Microbiology
University of Oklahoma School of Medicine
Oklahoma City

"Epidemiology of Sarcoidosis"

A clinical and epidemiologic review of 1194 Veteran patients with sarcoidosis. Previously shown at the National Tuberculosis Association meeting, the material is presented through the media of x-rays, photographs and graphs.

PHILIP C. JOHNSON, M.D.
Veterans Administration Hospital
Oklahoma City

"Use of Radioisotopes to Record Myocardial Blood Flow Changes Produced by Coronary Dilators"

The exhibit will demonstrate a new radioisotope technique that has been used to measure the changes in myocardial blood flow produced by coronary dilators in normal and arteriosclerotic hearts. The results obtained with this method are shown. Myocardial blood flow will be determined on those individuals desiring to be checked.

GILBERT L. HYROOP, M.D.
Oklahoma City

"Plastic and Reconstructive Surgery and Surgery of the Hand"

Photographs and x-rays of cases will be shown, both before and after plastic surgery.

OKLAHOMA STATE HEART ASSOCIATION
Oklahoma City

"Stop Rheumatic Fever: Techniques of Prevention"

This is an exhibit of diagnostic materials for the practicing physician to use in preventing rheumatic fever, such as throat culturing equipment, literature, models, photos and pamphlets.

Klinical Kaleidoscope

A Collection of Bizarre Cases: Located In The Center of The Scientific Exhibit Area.

R. KENT ALEXANDER, M.D.
Department of Medicine, Mercy Hospital
Oklahoma City
"Cushing's Disease and Actino Mycosis in a 30 Year Old Male"

TED CLEMENS, JR., M.D.
Oklahoma City
"Congenital Pancytopenia With Multiple Congenital Anomalies (Fanconi Type)"

JACK G. GLASGOW, M.D.
Oklahoma City
"Juvenile Rheumatoid Arthritis"

ONIS HAZEL, M.D.*
J. NEILL LYSAUGHT, M.D.
Oklahoma City
"Icthyosis Congenita, Due to Persistence of the Fetal Epitrichium"
*Deceased

ROBERT E. HERNDON, M.D.
Chickasha
"Malaria—Plasmodium Vivax"

MAXWELL A. JOHNSON, M.D.
Tulsa
"Giant Calculus In Accessory Urethra"

J. NEILL LYSAUGHT, M.D.
Oklahoma City
"Therapeutic Misadventure, or, When Is A Non-Poison A Poison?"

RENE B. MENGUY, M.D. and SAMUEL M. GLASSER, M.D.
Departments of Surgery and Radiology
Veterans Administration Hospital
Oklahoma City
"Acute and Chronic Pancreatitis"

BEN H. NICHOLSON, M.D.
Oklahoma City
"Congenital Absence of the Abdominal Musculature and Associated Anomalies"

PETER E. RUSSO, M.D.
Oklahoma City
"Congenital Lymphangioma"

E. A. McCUNE, M.D. and S. H. KOURI, M.D.
University of Oklahoma Medical Center
"Necrotizing Gastritis"

W. A. HEFLIN, M.D.
Ryan, Oklahoma
"Infantile Rheumatoid Arthritis"

W. T. McCOLLUM, M.D. and JOHN R. DANSTROM, M.D.
"Transposition of the Great Vessels"

JOHN M. MOORE, M.D.
Pauls Valley
"Methemoglobinemia"

GERALD BOLES, M.D., LOUIS SPEED, M.D., DAVID KRAFT, M.D., and WILLIAM SNODDY, M.D.
St. Anthony Hospital
Oklahoma City
"Adrenal Insufficiency Due to Histoplas... Capsulatum"

Relaxation . . .



Paul Neighbors

A must for Annual Meeting goers will be the fascinating music and sparkling entertainment of Paul Neighbors and his orchestra. Neighbors, a gifted emcee as well as a talented music man, will present a well-blended program of old and new, sweet and bouncy arrangements, designed to please the most particular admirers of good dance tempos and entertainment.

Sharing the spotlight with Paul will be the delightful Shirley Murray, bright young song stylist. Not to be out done in the song department, exuberant Neighbors will also take turns at the mike, as will "The Three Neighbors," a trio from his orchestra.

One of the nation's most talked about musical personalities, Paul Neighbors is sure to infect dancers and sitters-out alike with his charm and musical aplomb. Don't miss him!

President's Inaugural Dinner-Dance

TUESDAY, MAY 3, 1960

6:00 p.m. SOCIAL HOUR AND RECEPTION. Crystal and Balinese Rooms, Skirvin Hotel.

7:00 p.m. DINNER AND INAUGURAL CEREMONIES. Persian Room, Skirvin Tower Hotel.

9:00 p.m. DANCING to the music of Paul Neighbors, "The Prince of Personality" and his Orchestra. Persian Room, Skirvin Tower Hotel.

Ticket Information

Tickets to the President's Inaugural Dinner-Dance may be purchased in advance by writing: Oklahoma State Medical Association, Box 9696, Oklahoma City. The tickets, which are $7.50 each, will include the entire evening's entertainment: Social Hour, Dinner, Inaugural Ceremonies, Dance. Checks should accompany the order and should be made payable to the "Oklahoma State Medical Association."

Attendance to the Dinner-Dance is limited to the capacity of the Persian Room—So act now! If any tickets remain after mail orders have been filled, they will be sold at the Registration Desk in the Zebra Room, Municipal Auditorium.

. . . Recreation



Golf and Tennis Tournaments

Furnishing a perfect ending to Annual Meeting activities, the annual Golf and Tennis Tournaments of the Oklahoma State Medical Association will be held on Wednesday afternoon, May 4, at the Twin Hills Golf and Country Club and the Oklahoma City Tennis Club, respectively.

Golfers will tee off at 1:00 p.m. in groups of four. The rolling, wooded course, which is located at 3401 N.E. 36th Street, is one of the nation's finest, a favorite site for regional and national tournaments.

Following the play, a social hour and awards dinner will be held in the clubhouse. Among the trophies to be given are: Low Net, Handicap, Birdie Score, High Score, High Hole Score and Low Hole Score. Green fee for the tournament is $3.00 per person. The dinner is $3.50 each.

Tennis matches will also begin at 1:00 p.m. at the Oklahoma City Tennis Club courts, located adjacent to the Branding Iron Club, 5600 N. Portland Avenue.

Registration for both events will be taken at the general registration desk in the Zebra Room of the Municipal Auditorium.

Oklahoma State Medical Association

1959 DELEGATES AND ALTERNATES

COUNTY	DELEGATE	ALTERNATE
ATOKA	NO ENTITLEMENT	
BRYAN	B. B. Coker, M.D., Durant	Doyle Patton, M.D., Coalgate
COAL	NO ENTITLEMENT	
BECKHAM	NOT REPORTED	
BLAINE	NOT REPORTED	
CADDO	John H. Ennis, M.D., Cyril	A. Craig Roberson, M.D., Anadarko
CANADIAN	Jack Enos, M.D., Yukon	Alpha Johnson, M.D., El Reno
CARTER	NOT REPORTED	
LOVE	NO ENTITLEMENT	
MARSHALL	NO ENTITLEMENT	
COOKSON HILLS (Cherokee-Adair)	Bob G. Mitchell, M.D., Sallisaw	Brice Bliss, M.D., Tahlequah
CHOCTAW	Floyd L. Waters, M.D., Hugo	John D. Jennings, M.D., Hugo
PUSHMATAHA	NO ENTITLEMENT	
CLEVELAND	NOT REPORTED	
McCLAIN	NOT REPORTED	
COMANCHE	W. A. Matthey, M.D., Lawton	O. L. Parsons, M.D., Lawton
	Donald A. Angus, M.D., Lawton	J. T. Hicks, Jr., M.D., Lawton
COTTON	NO ENTITLEMENT	
CRAIG	Donald G. Olson, M.D., Vinita	
OTTAWA	W. G. Chesnut, M.D., Miami	
CREEK	Charles T. Kent, Jr., M.D., Bristow	Robert G. White, M.D., Sapulpa
CUSTER	NOT REPORTED	
EAST CENTRAL:		
MUSKOGEE	Francis R. First, Jr., M.D., Checotah	Joe James, M.D., Muskogee
	William Weaver, M.D., Muskogee	Bartis M. Kent, M.D., Muskogee
	John R. Rafter, M.D., Muskogee	George Kaiser, M.D., Muskogee
WAGONER	NO ENTITLEMENT	
SEQUOYAH	NO ENTITLEMENT	
McINTOSH	NO ENTITLEMENT	
GARFIELD	Henry T. Russell, M.D., Enid	Paul H. Rempel, M.D., Enid
	John W. Williams, M.D., Enid	Wilson J. Buvinger, M.D., Enid
	Avery B. Wight, M.D., Enid	H. L. Steffen, M.D., Enid
KINGFISHER	NOT REPORTED	
GARVIN	J. A. Graham, M.D., Pauls Valley	John M. Moore, M.D., Pauls Valley
GRADY	Joseph J. Swan, M.D., Chickasha	J. H. Foertsch, M.D., Chickasha
GRANT	NO ENTITLEMENT	
GREER	NOT REPORTED	
HUGHES	NOT REPORTED	
SEMINOLE	NOT REPORTED	
JACKSON	Wayne A. Starkey, M.D., Altus	Robert S. Srigley, M.D., Altus
JEFFERSON	NOT REPORTED	
KAY	J. M. Bush, M.D., Ponca City	Harold Jones, M.D., Ponca City
	L. H. Becker, M.D., Blackwell	R. F. Morgan, M.D., Backwell
NOBLE	Arthur Brown, M.D., Perry	Charles E. Martin, M.D., Perry
KIOWA	William Bernell, M.D., Hobart	J. B. Tolbert, M.D., Mountain View
WASHITA	A. E. Stowers, M.D., Sentinel	L. G. Livingston, M.D., Cordell
LeFLORE	NOT REPORTED	
HASKELL	NO ENTITLEMENT	
LINCOLN	Ned Burleson, M.D., Prague	Harold T. Baugh, M.D., Meeker
LOGAN	L. H. Ritzhaupt, M.D., Guthrie	James S. Petty, M.D., Guthrie
McCURTAIN	NOT REPORTED	
MURRAY	Ralph W. Morton, M.D., Sulphur	Jack W. Donald, M.D., Sulphur
NORTHWEST:		
BEAVER	NO ENTITLEMENT	
DEWEY	NO ENTITLEMENT	

ELLIS	M. H. Newman, M.D., Shattuck	R. H. Burgtorf, M.D., Shattuck
HARPER	NO ENTITLEMENT	
WOODWARD	E. A. McGrew, M.D., Beaver	R. G. Obermiller, M.D., Woodward
OKFUSKEE	NOT REPORTED	
OKLAHOMA		
	H. T. Avey, M.D.	Robert M. Bird, M.D.
	Irwin H. Brown, M.D.	J. N. Lysaught, M.D.
	C. Alton Brown, M.D.	Chas. M. Harvey, M.D.
	Thomas C. Points, M.D.	Kenneth G. Ogg., M.D.
	Jim M. Taylor, M.D.	John A. Cunningham, M.D.
	Ira O. Pollock, M.D.	J. M. White, Jr., M.D.
	Mark R. Johnson, M.D.	Arthur Schmidt, M.D.
	Robert C. Lawson, M.D.	Elmer R. Musick, M.D.
	Lynn H. Harrison, M.D.	Alvin R. Jackson, M.D.
	Ella H. Murray, M.D.	James S. Boyle, M.D.
	Vernon D. Cushing, M.D.	John W. DeVore, M.D.
	Virgil Ray Forester, M.D.	J. J. Gable, M.D.
	V. M. Rutherford, M.D.	Robert J. Morgan, M.D.
	E. E. Shircliff, M.D.	Chas. E. Delhotal, M.D.
	P. D. Casper, M.D.	Dick Lowry, M.D.
	Jack G. Glasgow, M.D.	John F. Kuhn, M.D.
	E. Cotter Murray, M.D.	M. Joe Crosthwait, M.D.
	Paul D. Macrory, M.D.	Elwood Herndon, M.D.
	A. C. Lisle, Jr., M.D.	Arnold G. Nelson, M.D.
	Richard E. Carptenter, M.D.	M. T. Buxton, Jr., M.D.
	Robert T. Sturm, M.D.	E. Stanley Berger, M.D.
	S. R. Shaver, M.D.	M. B. Glismann, M.D.
OKMULGEE	Carlton E. Smith, M.D., Henryetta	Cleve Beller, M.D., Okmulgee
OSAGE	William Geiger, Jr., M.D., Fairfax	Vincent Mazzarella, M.D., Hominy
PAYNE	George R. Smith, M.D., Cushing	D. W. Humphreys, M.D., Cushing
PAWNEE	R. D. Hargrove, M.D., Pawnee	M. L. Saddoris, M.D., Cleveland
PITTSBURG	Thurman Shuller, M.D., McAlester	G. M. Brown, Jr., M.D., McAlester
	F. T. Bartheld, M.D., McAlester	E. D. Greenberger, M.D., McAlester
PONTOTOC	NOT REPORTED	
POTTOWATOMIE	Frances P. Newlin, M.D., Shawnee	Francis A. Davis, M.D., Shawnee
ROGERS	NOT REPORTED	
MAYES	NOT REPORTED	
STEPHENS	W. R. Cheatwood, M.D., Duncan	W. K. Walker, M.D., Marlow
TEXAS		
CIMARRON	NO ENTITLEMENT	
TILLMAN	Roger G. Johnson, M.D., Frederick	William G. Harvey, M.D., Frederick
TULSA		
	Hugh Perry, M.D.	James B. Thompson, M.D.
	John E. McDonald, M.D.	Byron W. Steele, Jr., M.D.
	Walter S. Larrabee, M.D.	William R. Turnbow, M.D.
	Simon Pollack, M.D.	Robert T. Cronk, M.D.
	Harold J. Black, M.D.	Worth M. Gross, M.D.
	James C. Peters, M.D.	Gerald E. Cronk, M.D.
	Charles E. Wilbanks, M.D.	Craig S. Jones, M.D.
	Earl M. Lusk, M.D.	William C. Alston, Jr., M.D.
	Paul O. Shackelford, M.D.	Homer D. Hardy, Jr., M.D.
	N. C. Gaddis, M.D.	Dixon N. Burns, M.D.
	Ben F. Gorrell, M.D.	Richard E. McDowell, M.D.
	F. L. Flack, M.D.	G. R. Krietmeyer, M.D.
	Vincel Sundgren, M.D.	Milton L. Berg, M.D.
	Rayburne W. Goen, M.D.	Byron L. Bailey, M.D.
WASHINGTON	Fred C. Wallingford, M.D., Bartlesville	Clair Liebrand, M.D., Bartlesville
	C. S. Huntington, M.D., Bartlesville	H. E. Denyer, M.D., Bartlesville
NOWATA	Lynn C. Barnes, M.D., Nowata	O. L. Grigsby, M.D., Nowata
WOODS	John X. Blender, M.D., Cherokee	John Simon, M.D., Alva
	Forrest Hale, M.D., Cherokee	T. D. Benjergerdes, M.D., Alva

Other Groups to Meet

(Those marked with an asterisk are open to all O.S.M.A. members)

American Academy of Pediatrics, Oklahoma Chapter

The Oklahoma Chapter of the American Academy of Pediatrics will meet on Monday, May 2, in the Venetian Room of the Skirvin Hotel. A social hour will begin at 6:30 p.m., followed by a dinner, business meeting and lecture by Murdina M. Desmond, M.D., Houston. Doctor Desmond's subject will be "The Infants of Mothers Addicted to Narcotics." Tickets for the entire evening are $5.00 each.

*American College of Obstetricians and Gynecologists, Oklahoma Section

College members and interested OSMA members will meet on May 2, in the Crystal Room of the Skirvin Hotel, beginning at 3:45 p.m., John W. Records, M.D., presiding. Jed E. Goldberg, M.D., Tulsa, will speak on "Renal Tubular Acidosis in Pregnancy," and Gerald Rogers, M.D., Oklahoma City, will deliver a paper entitled "Treatment of Post-Operative Vesicovaginal Fistula (with Report of 28 Consecutive Cures)." Schuyler Kohl, M.D., Brooklyn, will discuss both papers.

At 6:30 p.m., a social hour will be held for physicians and their wives, followed by dinner at 7:30. Tickets are $7.50 each. Following dinner, Doctor Kohl will address the group on "Early Discharge (48 hours) of Obstetrical Patients."

American College of Surgeons, Oklahoma Chapter

American College of Surgeons members and their wives will meet on Monday, May 2, in the Continental Room of the Skirvin Hotel. The evening will begin with a social hour at 7:00 p.m., followed by a dinner at 8:00 p.m. Tickets are $10.00 each.

*American Psychiatric Association, Oklahoma District Branch

Oklahoma members of the American Psychiatric Association and other interested members of the OSMA will meet on Monday, May 2, at the Biltmore Hotel. APA members will have a business meeting at 5:00 p.m. The open meeting will begin with a 6:00 p.m. social hour, and will conclude with a 7:00 p.m. dinner. Guest speaker for the dinner will be Harold Rosen, M.D., Baltimore, Maryland.

*Oklahoma Orthopedic Society

The orthopedic surgery group will meet twice during the annual meeting.

On Sunday, May 1, the Society members will meet at Crippled Children's Hospital at 2:00 p.m. for case presentations and discussion by Harold A. Sofield, M.D., Oak Park, Illinois. A social hour and dinner is also scheduled at 5:30 p.m. at Glen's Hick'ry Inn.

Monday evening, May 2, the orthopedists will host other members of the OSMA in the Monterey Room of the Skirvin Hotel.

A panel, "Orthopedic Problems in the Office," will begin at 4:00 p.m., and will feature Doctor Sofield. At 5:30 p.m. there will be a social hour, and, at 6:30 p.m., a dinner. Tickets for members and non-members are $6.50 each.

Oklahoma State Dermatological Society

Members of the state dermatological group are scheduled to meet on May 2. From 3:00 p.m. to 5:00 p.m., case presentations and comments will be made at the office of John H. Lamb, M.D. At 6:00 p.m., a social hour will be held at the Faculty House, 601 N.E. 14th, followed by dinner at 7:00 p.m. Vincent Derbes, M.D., New Orleans, will speak to dermatologists and allergists at the dinner session.

Oklahoma State Internist Association

The Oklahoma State Internist Association will meet at the Skirvin Hotel on May 2. A social hour is set for 6:15 p.m. and a dinner for 7:00 p.m. Following dinner, Edgar V. Allen, M.D., Rochester, Minnesota, will present "Some Remarks on Atheromatosis." At approximately 8:40 p.m., the association will conduct its annual business meeting.

Oklahoma State Radiological Society

The Executive Suite, Skirvin Hotel, will be the site of the radiology meeting on Monday, May 2. A social hour is scheduled for 6:30 p.m., and a dinner for 7:00 p.m. Benjamin Felson, M.D., Cincinnati radiologist, will speak on "Splenoportography."

Oklahoma State Urological Society

Oklahoma urologists will meet on Saturday, April 30, from 9:30 a.m. until 5:00 p.m. at the Skirvin Hotel. Grayson Carroll, M.D., St. Louis, will lecture on "Selection of Drugs in Resistant Urinary Infections: Colymycin—Kanamycin—Triacetyloleandomycin."

PLAN TO ATTEND THE 109th ANNUAL MEETING OF THE AMERICAN MEDICAL ASSOCIATION.

June 13 -17, 1960, Miami Beach, Florida

Write Convention Services, American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois for advance meeting registration information

For The Ladies . . .



MRS. CLIFFORD M. BASSETT
Cushing
President
Woman's Auxiliary
Oklahoma State
Medical Association



MRS. VIRGIL RAY FORESTER
Oklahoma City
President-Elect
Woman's Auxiliary
Oklahoma State
Medical Association



MRS. WILLIAM MACKERSIE
Detroit, Michigan
President-Elect
Woman's Auxiliary
American
Medical Association



MRS. JOHN M. CHENAULT
Decatur, Alabama
President
Woman's Auxiliary
Southern
Medical Association

STATE OFFICERS



MRS. T. A. RAGAN
Norman
1st Vice-President



MRS. MILTON L. BERG
Tulsa
2nd Vice-President



MRS. CHARLES W. FREEMAN
Oklahoma City
Secretary



MRS. JOHN W. RECORDS
Oklahoma City
Treasurer



MRS. C. F. FOSTER, Jr.
Oklahoma City
General Chairman

WOMAN'S AUXILIARY

to the

OKLAHOMA STATE MEDICAL ASSOCIATION

ANNUAL MEETING

May 1 - 4, 1960

SKIRVIN HOTEL, OKLAHOMA CITY, OKLAHOMA

MRS. C. F. FOSTER, Jr.
Convention Chairman

REGISTRATION AND INFORMATION

Sunday, May 1

Mezzanine, Skirvin Hotel—3:00 p.m.-5:00 p.m.

Monday and Tuesday, May 1 and 2

Foyer, 14th floor, Skirvin Hotel—9:00 a.m.-12:00 noon

HOSPITALITY ROOM

The Hospitality Room will be open during registration hours Sunday, Monday and Tuesday for the convenience of members and guests. Refreshments will be served.

OKLAHOMA DOCTORS' HOBBY SHOW

MRS. WILLIAM R. R. LONEY,
Chairman

MRS. HARRELL C. DODSON,
Co-Chairman

Hobbies of Oklahoma Physicians are presented in this interesting exhibit and will be on display in the Regency Room, Mezzanine, The Skirvin Hotel.

TICKETS

Tickets for the various luncheons will be sold at the Registration desks.

. . . A Full Schedule

PROGRAM

SUNDAY, MAY 1, 1960

1:00 p.m.—EXECUTIVE BOARD MEETING AND LUNCHEON, Balinese Room, Skirvin Hotel.

3:00 p.m.-5:00 p.m.—REGISTRATION AND INFORMATION, Mezzanine, Skirvin Hotel.

MONDAY, MAY 2, 1960

8:30 a.m.—PAST PRESIDENTS' BREAKFAST, Blue Room, Skirvin Hotel. Hostess: Mrs. John Powers Wolff.

9:00 a.m.—REGISTRATION AND INFORMATION, Foyer of 14th Floor, Skirvin Hotel. Coffee and Rolls will be served.

10:00 a.m.—GENERAL MEETING, Venetian Room, 14th Floor, Skirvin Hotel. Mrs. Clifford M. Bassett, President, Woman's Auxiliary to to the Oklahoma State Medical Association, presiding.

CALL TO ORDER: Mrs. Bassett.

INVOCATION: Mrs. Milton Berg, 2nd Vice-President.

PLEDGE OF LOYALTY: Mrs. T. A. Ragan, 1st Vice-President.

"I pledge my loyalty and devotion to the Woman's Auxiliary to the American Medical Association. I will support its activities, protect its reputation and ever sustain its high ideals."

WELCOME: Mrs. Joseph J. Maril, President, Oklahoma County Medical Auxiliary.

GREETINGS: Walter E. Brown, M.D., President-Elect, Oklahoma State Medical Association.

Mrs. Elias Margo, 2nd Vice-President, Woman's Auxiliary to the Southern Medical Association.

GUEST-SPEAKER: Mrs. William Mackersie, President-Elect, Woman's Auxiliary, American Medical Association.

IN MEMORIAM: Mrs. Virgil Ray Forester, President-Elect, Woman's Auxiliary to the Oklahoma State Medical Association, assisted by Mrs. H. V. L. Sapper and Mrs. G. S. Collins, Jr.

ANNOUNCEMENTS BY GENERAL CHAIRMAN: Mrs. C. F. Foster, Jr.

ROLL CALL BY COUNTIES: Mrs. John W. Records, Treasurer, Woman's Auxiliary to the Oklahoma State Medical Association.

TREASURER'S REPORT: Mrs. Records.

READING AND ADOPTION OF THE MINUTES: Mrs. Charles W. Freeman, Secretary, Woman's Auxiliary to the Oklahoma State Medical Association.

REPORT OF THE CREDENTIALS COMMITTEE: Mrs. Coye W. McClure.

REPORTS AND INTRODUCTION OF OFFICERS AND CHAIRMEN:

First Vice-President, Mrs. T. A. Ragan
Second Vice-President, Mrs. Milton L. Berg
Corresponding Secretary, Mrs. Louis S. Frank
Parliamentarian, Mrs. Iron Hawthorne Nelson
Historian, Mrs. George H. Garrison
Editor, Mrs. Samuel T. Moore
Co-Editor, Mrs. John Powers Wolff
American Medical Education Foundation, Mrs. Pat Fite, Sr.
Civil Defense, Mrs. J. F. York
Community Service, Mrs. Tom C. Sparks
Doctor's Day, Mrs. Lawrence E. Thompson
Doctor's Hobby, Mrs. Wm. R. R. Loney
Co-Chairman, Mrs. Harrell C. Dodson, Jr.
Legislation, Mrs. E. Clyde Mohler
Loan Fund, Mrs. Wm. R. Cheatwood
Mental Health, Mrs. George H. Guthrey
National Bulletin, Mrs. Peter A. MacKercher
Paramedical Careers, Mrs. H. C. Manning
Program-Health Education, Mrs. James P. Luton
Safety, Mrs. Floyd Bartheld
Student American Medical Auxiliary, Mrs. Robert J. Morgan
Hospitality, Mrs. Joseph W. Funnell
Co-Chairman, Mrs. John A. Blaschke
Revisions and Resolutions, Mrs. Clinton F. Gallaher
Finance-Budget, Mrs. John A. Cunningham

REPORT OF THE NOMINATING COMMITTEE, Mrs. Virgil Ray Forester, President Elect, Woman's Auxiliary to the Oklahoma State Medical Association.

ANNOUNCEMENTS

ADJOURNMENT

1:00 p.m.—LUNCHEON, FASHION SHOW, Persian Room, Skirvin Tower, in honor of Mrs. Clifford M. Bassett, President, and Mrs. Virgil Ray Forester, President-Elect of Woman's Auxiliary to the Oklahoma State Medical Association.

SPECIAL GUESTS: Mrs. William Mackersie, President-Elect, Woman's Auxiliary to the American Medical Association, Mrs. Neil Woodward, Civil Defense Chairman, Woman's Auxiliary to the American Medical Association, Mrs. E. Clyde Mohler, Area Legislative Chairman, Woman's Auxiliary to the American Medical Association, and Mrs. Elias Margo, 2nd Vice-President, Woman's Auxiliary to the Southern Medical Association.

FASHIONS BY KERR'S

JEWELS BY HARRY WINSTON, Inc., New York

TUESDAY, MAY 3, 1960

9:00 a.m.—REGISTRATION AND INFORMATION, Foyer, 14th Floor, Skirvin Hotel, Coffee and Rolls will be served.

10:00 a.m.—GENERAL MEETING: Venetian Room, 14th Floor, Skirvin Hotel, Mrs. Clifford M. Bassett, President, Presiding.

CALL TO ORDER: Mrs. Bassett.

INVOCATION: Mrs. E. Clyde Mohler, Past President, Woman's Auxiliary to The Oklahoma State Medical Association.

PLEDGE OF LOYALTY: Mrs. Iron H. Nelson, Past President, Woman's Auxiliary to the Oklahoma State Medical Association.

"I pledge my loyalty and devotion to the Woman's Auxiliary to the American Medical Association. I will support its activities, protect its reputation and ever sustain its high ideals."

WELCOME: Mrs. J. W. Funnell, President-Elect, Oklahoma County Medical Auxiliary.

GREETINGS: Alfred T. Baker, M.D., President, Oklahoma State Medical Association.

Mrs. John M. Chenault, President, Woman's Auxiliary to the Southern Medical Association.

INTRODUCTIONS:

ROLL CALL BY COUNTIES: Mrs. John W. Records, Treasurer, Woman's Auxiliary to the Oklahoma State Medical Association.

REPORTS: COUNTY PRESIDENTS:

Atoka-Bryan-Coal ___ Mrs. Seals L. Whitely
Carter-Love-Marshall _____ Mrs. Clifford Lorentzen
Cleveland-McClain ____ Mrs. H. W. Hackler
Comanche _____ Mrs. O. L. Parsons
Craig-Ottawa _____ Mrs. Donald Olson
East Central _____ Mrs. J. Hutchings White
Garfield-Kingfisher _____ Mrs. E. W. Bank
Grady-Caddo _____ Mrs. John Ennis
Jackson-Harmon _____ Mrs. Cooper D. Ray
Kay-Noble _____ Mrs. V. C. Merrifield
Kiowa-Washita _____ Mrs. Aubrey Stowers
Lincoln-Pottawatomie ____ Mrs. Claude Hirst
Oklahoma _____ Mrs. Joseph J. Maril
Okmulgee _____ Mrs. Charles S. Maben
Payne-Pawnee ____ Mrs. Geo. B. Gathers, Jr.
Pittsburg _____ Mrs. Bruce H. Brown
Pontotoc-Johnson-Murray _____ Mrs. Ollie McBride
Rogers-Mayes _____ Mrs. Robert M. Stover
Stephens _____ Mrs. Robert Taylor
Tulsa _____ Mrs. James W. Kelley
Washington-Nowata _____ Mrs. S. G. Weber

REPORT OF CREDENTIALS COMMITTEE: Mrs. Coye W. McClure.

REPORT OF FINANCE AND BUDGET COMMITTEE: Mrs. John Cunningham.

OLD BUSINESS

NEW BUSINESS

ELECTION OF DELEGATES TO NATIONAL CONVENTION

ELECTION OF OFFICERS

INSTALLATION OF OFFICERS: Mrs. George H. Garrison.

PRESENTATION OF PAST PRESIDENT'S EMBLEM: Mrs. Garrison.

PRESENTATION OF PRESIDENT'S PIN AND GAVEL: Mrs. Bassett.

ANNOUNCEMENTS: Mrs. C. F. Foster, Jr.

ADJOURNMENT:

1:00 p.m.—LUNCHEON—POST SCHOOL OF INSTRUCTION, Continental Room, 14th Floor, Skirvin Hotel, Mrs. Virgil Ray Forester, Presiding.

6:00 p.m.—SOCIAL HOUR, Balinese and Crystal Rooms, Skirvin Hotel.

7:00 p.m. PRESIDENT'S INAUGURAL DINNER DANCE, Persian Room, Skirvin Tower.

CONVENTION COMMITTEES

MRS. C. F. FOSTER, Jr., *Chairman*

Registration	Mrs. Robert Ellis
	Mrs. Richard Clay
Credentials	Mrs. Coye W. McClure
Courtesy	Mrs. Joseph W. Funnell
	Mrs. John A. Blaschke
Hospitality	Mrs. Marshall Opper
	Mrs. E. Cotter Murray
Hobby Show	Mrs. Wm. R. R. Loney
	Mrs. Harrell C. Dodson
Luncheon and	Mrs. Gerald Bednar
Fashion Show	Mrs. Henry C. Traska
	Mrs. Dick Huff
Past Presidents	
Breakfast	Mrs. John Powers Wolff
Tickets	Mrs. Arthur F. Elliott
	Mrs. Jesse E. Miller
Publicity	Mrs. Virgil Ray Forester

Oklahoma State Medical Assistants Society

Follow . . . "HIGHWAYS to HARMONY" to the

12th Annual Meeting of the

OKLAHOMA STATE MEDICAL ASSISTANTS SOCIETY

April 29, 30-May 1, 1960 Hotel Biltmore
Oklahoma City

FRIDAY, APRIL 29

7:30- 8:30 p.m.—Registration

8:30-10:30 p.m.—Reception
Hostess: Garfield-Kingfisher Medical Assistants
Enid, Oklahoma

SATURDAY, APRIL 30—MORNING SESSION

8:00-9:00 a.m.—Registration, French Lounge

9:00 a.m.—Call to order Derrick Room
Presiding: NENA CHADSEY, President, Oklahoma State Medical Assistants Society, Bartlesville, Oklahoma

Invocation, Mary Rice, Chaplain, Oklahoma City
Welcome, Honorable James Norick, Mayor of Oklahoma City

Response, Mrs. Lillian Lewellen, Henryetta, Oklahoma

Official business

11:20 a.m.—Guest Speaker, Joan Barlow, R.N., E. R. Squibb Co., "How Squibb can help Medical Assistants"

Noon—Luncheon, Montego Room
"Civil Defense," Mr. Tom Brett, Director, Oklahoma Civil Defense

Hostess: Comanche County Medical Assistants
Lawton, Oklahoma

AFTERNOON SESSION

2:00 p.m.—Business Session resumed, Derrick Room. Roll Call, Recognition of Delegates, Election of Officers—Nena Chadsey, Presiding.

3:00 p.m.—Oklahoma Blue Cross-Blue Shield, Mr. Carl Behle, Director, Professional Relations.

6:00-7:00 p.m.—Cocktails—Hosts: Medical Service Society of Oklahoma City.

7:00 p.m.—Banquet, Montego Room.

Hostess: Tulsa County Medical Assistants
Greetings, Leo J. Starry, M.D., Oklahoma City, (Life Member, A.A.M.A.)

Guest Speaker, "Anecdotes," Francis W. Pruitt, M.D., Tulsa, Oklahoma, Coordinator Graduate Education, Tulsa Hillcrest Medical Center; former Chief Dept. Med., Walter Reed Army Hospital and Medical Consultant to the White House; Brig. Gen.—USA,MC retired.

9:30-12:30—DANCING, "Good Music"—Al Good and Orchestra

Hostess: Oklahoma County Medical Assistants,
Oklahoma City

SUNDAY, MAY 1—MORNING SESSION

7:30-8:30 a.m.—A la Carte Breakfast, Balcony, Coffee Shop

Hostess: Washington-Nowata County, Medical Assistants, Bartlesville, Oklahoma

9:00 a.m.—Business Session resumed, Derrick Room, County Reports.

10:00 a.m.—"Office Techniques and the Happy Patient," Johnny A. Blue, M.D., Oklahoma City, Secretary-Treasurer, Oklahoma State Medical Society.

Hostess: Washington-Nowata County Medical Assistants

Noon—Luncheon, Mirror Room.
"Fashion Trends in the Practice of Medicine,"
Port Johnson, M.D., Muskogee Medical Center.

Hostess: East Central Oklahoma Medical Assistants, Muskogee, Oklahoma

AFTERNOON SESSION

1:30 p.m.—Installation of New Officers, Lucille Swearingen, Jr. Past President A.A.M.A., Bartlesville
Courtesy Resolutions

Committee Appointments and Address by New President, Gertrude Habiger, Ponca City, Oklahoma.

2:30 p.m. "I Am a Doctor," Color Film—American Medical Association.

Hostess: Oklahoma County Medical Assistants

3:00 p.m.—Awards and adjournment.

General Convention Chairman
Gerry Schwarz—No. 10—525 N.W. 11
Oklahoma City, Oklahoma

Registration Fee: (includes two receptions, lunch and dinner-dance on Saturday, breakfast and lunch Sunday)—Members $12.50; Others $17.50.

Fifty-Five Technical Exhibitors Scheduled

The Technical Exhibit at the 54th Annual Meeting of the Oklahoma State Medical Association will feature displays by the following leading firms:

Exhibitors

Abbott Laboratories
American Association of Physicians and Surgeons
Blue Cross and Blue Shield Plans
Burroughs Corporation
Burt's Business Machines
Ciba Pharmaceutical Products, Inc.
Coca-Cola Company
Connie's Prescription Shop
Credit Service
Dictaphone Corporation
Doho Chemical Corporation
Electronic Business Machines, Inc.
Endo Laboratories
Flint-Eaton and Co.
Geigy Pharmaceuticals
General Electric Company
Greb X-Ray Company
J. E. Hanger & Company
The G. F. Harvey Company, Inc.
Lederle Laboratories
Eli Lilly Company
Lloyd Brothers, Inc.
J. A. Majors Company
Marion Laboratories, Inc.
The S. E. Massengill Company
Mead Johnson & Company
Medco Products Company, Inc.
Melton Company, Inc.
Melton-Myers, Inc.
Mid-Continent Surgical Supply Co.
Midwest Surgical Supply Company, Inc.
Merrill, Lynch, Pierce, Fenner & Smith
V. Mueller & Company
The North American Company for Life, Accident and Health Insurance
Oklahoma Physicians Supply, Inc.
Ortho Pharmaceutical Corporation
Parke, Davis and Company
Pipkin Photo Service
Purdue-Frederick Company
R. J. Reynolds Company
Sabolich Orthopedic Appliance Company
Sandoz Pharmaceuticals
Schering Corporation
Sealy Southwest
G. D. Searle & Company
7-Up Bottlers of Oklahoma
Smith, Miller and Patch, Inc.
St. Paul Mercury Insurance Company
E. R. Squibb & Sons
S. J. Tutag and Co.
U. S. Vitamin Corporation
Warren-Teed Products Company
Westwood Pharmaceutical Company
Winthrop Laboratories
Wyeth Laboratories

News

Forand Threat Eases; More Trouble Ahead

A momentary victory was achieved by anti-Forand forces on March 23 when the House Ways and Means Committee heard representatives of the Administration reaffirm opposition to government intervention into the health care field. The effect of the testimony was to virtually kill the bill as far as the committee is concerned, but new threats are now looming on the horizon.

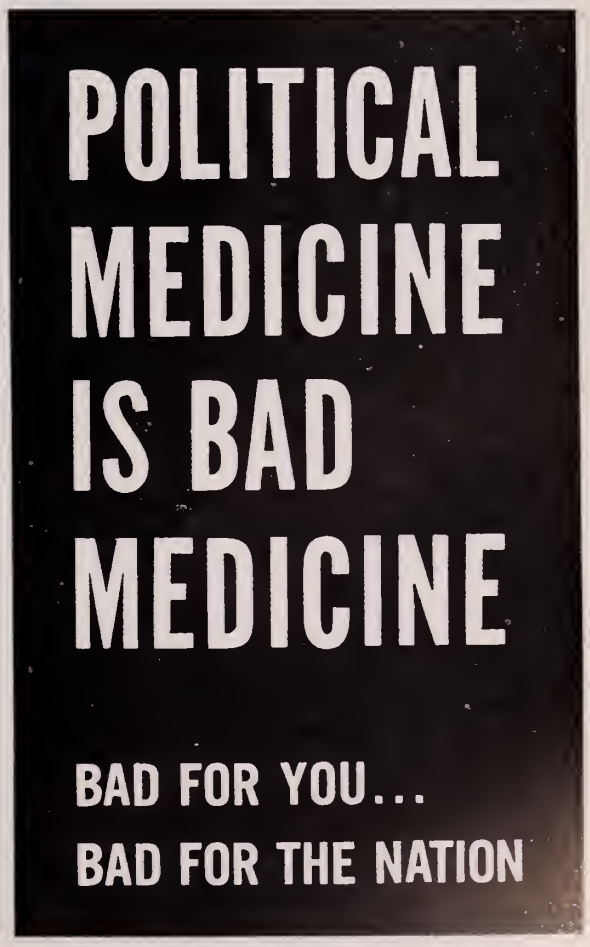
Prior to the time Arthur S. Fleming, Secretary of Health, Education and Welfare, appeared before the committee on behalf of the Administration, the fate of the Forand Bill was in the hands of the Republican members of the committee who were successfully blocking any action. Shortly before the testimony of Flemming, however, it was reported that he and the liberal wing of the Republican Party were working on Forand-Type legislation to offer as a counter proposal. This development was met by a stepped up writing campaign on the part of physicians and other anti-socialized medicine groups. Opponents of the Forand Bill were rewarded when Fleming reluctantly reported the Administration's opposition to Forand-type legislation, thus giving support to the conservative members of the committee who have steadfastly refused to yield to pressure from the AFL-CIO and other groups.

With the Administration's reaffirmation of opposition, the forand Bill, H. R. 4700, has little chance of being reported out of committee this session of Congress. Proponents of the legislation are now changing their tactics, and the threat of H. R. 4700 or similar legislation is still imminent.

Representative Aime Forand is now circulating H. R. 483, a discharge petition which will bring H. R. 4700 directly to the floor of the Houe for a vote, provided he can get 219 Representatives to sign it. In addition to this political maneuvering, it is quite probable that Senators Kennedy and Humphrey will try to attach Forand-type amendments to the Social Security bill when it reaches the floor of the Senate.

Kennedy has already launched one effort in this direction when he recently introduced a bill which would provide hospital and nursing care for Social Security beneficiaries (in contrast to Forand's which would provide *medical, dental,* hospital and nursing home care).

Oklahoma physicians and other free enterprisers must maintain a continuous letter-writing campaign to their Representatives and Senators. Representatives should be asked to refrain from signing the discharge petition and Senators Kerr and Monroney should be admonished to work against any Forand-type legislation originating in the Senate.



Individual physicians may order this free pamphlet from the AMA, 535 North Dearborn Street, Chicago 10, Illinois.

Miami Beach Offers Ideal Climate, Diverse Activities for AMA Meeting

Miami Beach, a lush chain of islets set in the sparkling Atlantic, promises ideal weather and vast entertainment facilities for the 109th annual meeting of the American Medical Association, June 13-17.



The meeting is AMA's first in Miami Beach since the winter of 1954.

The fabled resort area offers the ultimate in sun and fun—temperatures averaging from 75 to 85 in June and a variety of activities to fit every taste.

Miami Beach escapes the extremes of heat and cold by virtue of the warm waters of the Gulf Stream and its peninsular location. In summer, the sun is tempered by fleecy clouds and occasional rain.

The beauty of the city is enhanced by the trees, shrubs and flowers that bloom in abuandance in private gardens and public parks.

An estimated 125,000 visitors can be accommodated in Miami Beach at any given time. Including metropolitan Miami, there are a total of 917 hotels and motels with 55,600 rooms.

The waters of the Atlantic and Biscayne Bay offer ample opportunity for swimming, skiing, skin diving and fishing.

There are miles of beaches dotted with palms, water ski schools and coral reefs for skin divers to explore. Sail boats and motor boats may be rented.

Fishing boats also are available for charter. One of the most popular sports is Gulf Stream trolling for sailfish, marlin and other salt-water heavyweights. Freshwater fishermen will find game fish in bay and inlet waters, such as tarpon from 10 to 150 pounds or more.

Sightseers can enjoy a breathtaking view of scenic residential islands and waterfront estates from the deck of modern cruisers that ply the bay. A glass bottom sightseeing boat, "The Mermaid," also operates from the City Yacht Basin.

For golfers, there are two championship courses in Miami Beach, 12 others in the city of Miami.

Shopping is a pleasant pastime along palm-lined Lincoln Road stretching from the bay to the ocean. Many of the shops are branches of world-famous houses.

For nighttime diversion, Miami Beach features some of the nation's top stars and revues at smart supper clubs along with many fine restaurants. Summer menus usually include fish served fresh from the water.

Among the interesting places to go in the Miami area is Vizcaya, a magnificent 16th century Italian palazzo which houses the Dade County Art Museum.

The 69-room mansion contains a priceless collection of European and Asiatic art from the first to the 19th century. It is surrounded by 10 acres of formal gardens.

Nearby islands within easy reach of Miami include Cuba, Haiti, Jamaica, Nassau, Puerto Rico and others in the West Indies. Most are a short air trip or an overnight boat trip from the mainland. It's possible to go to Cuba by car. From Key West, an auto ferry makes the trip in seven hours.

Tulsans Hear AMA Leader

E. Vincent Askey, M.D., of Los Angeles, President-Elect of the American Medical Association, said last month that the Senior Citizens Insurance Program recently approved by the Oklahoma State Medical Association was "the sensible and practical way of providing medical care to the aged."

Speaking at the March 14th dinner meeting of the Tulsa County Medical Society to an audience of 260 doctors and wives, the California surgeon attacked the "ruinous taxation" as a major reason for defeat of the Forand Bill and similar types of legislation.

Doctor Askey praised the action of the OSMA House of Delegates in adopting a Blue Cross-Blue Shield policy for sale to over-60 citizens. "This type of leadership can provide the means of effectively combating the Forand Bill, yet providing our Social Security beneficiaries and other aged persons with the best medical care which a free and unregulated (by government) profession can offer."

Following the dinner meeting, Doctor Askey was presented with gifts from the Tulsa County Medical Society and the Oklahoma State Medical Association. A telegram of congratulations from Governor J. Howard Edmondson of Oklahoma, praising Doctor Askey for his leadership in medical circles was also read.

Guests at the event included Alfred T. Baker, M.D., President of the Oklahoma State Medical Association, Durant; Malcom E. Phelps, M.D., AMA Delegate, El Reno; Wilkie D. Hoover, M.D., AMA Delegate, Tulsa; R. Q. Goodwin, M.D., AMA Alternate Delegate, Oklahoma City; W. A. Showman, M.D., AMA Delegate from the Section on Dermatology; Robert T. Sturm, M.D., President of the Oklahoma Chapter of the American Academy of General Practice, Oklahoma City; A. C. Lisle, jr., M.D., President of the Oklahoma County Medical Society; Mrs. Alma F. O'Donnell, Executive Secretary of the Oklahoma County Medical Society; and Walter E. Brown, M.D., Tulsa, President-Elect of the Oklahoma State Medical Association.

Heart Patient Course and Special Seminars Planned

The Heart Patient, a short course sponsored by the Oklahoma State Heart Association, the Oklahoma State Department of Health and the University of Oklahoma Medical Center, Extension Division, has been planned in connection with Special Seminars for physicians, April 18-29, 1960 at the University of Oklahoma Medical Center.

Registration fee for the course is $25.00 (with or without credit) for all or any portion of the course. Tuition scholarships are available.

Two hours credit in Preventive Medicine and Public Health 221 will be allowed for those who are eligible. In addition, members of the American Academy of General Practice may receive a maximum of 37½ hours credit acceptable for category II. Attendance at one or more of the seminars will be given proportional credit.

Inquiries may be addressed to the Oklahoma Heart Association, 825 Northeast 13th, Oklahoma City.

A complete program follows:

FIRST WEEK

MONDAY, APRIL 18

INTRODUCTION

8:30-10:00 a.m. Registration
10:15-10:20 a.m. Welcome to Course—Dean Mark R. Everett
10:20-10:30 a.m. Introduction—Doctor Herbert B. Hudnut, Jr.
10:30-10:45 a.m. "Care of the Patient with Heart Disease"—Doctor Stewart G. Wolf, Jr.
11:45-12:15 p.m. Flim—Discussion
12:15- 1:15 p.m. Lunch
1:15- 2:00 p.m. "The Heart and Circulation"—Doctor A. N. Taylor
2:00- 3:00 p.m. "Major Heart Diseases"—Doctor William E. Jacques
3:15- 4:15 p.m. Diets and Heart Disease"—Miss Beth Heap
4:15- 5:00 p.m. Film—Discussion

TUESDAY, APRIL 19

PROBLEMS OF THE PATIENT WITH CORONARY ARTERY DISEASE

8:30- 9:30 a.m. "Problems of Patients with Coronary Artery Disease"—Doctor William Best Thompson
9:30-10:00 a.m. Discussion
10:15-12:15 p.m. Case Presentation—Panel Discussion

12:15- 1:15 p.m. Lunch
1:15- 3:15 p.m. Special seminars for physicians*
4:00- 5:00 p.m. Lecture, "Serum Lipids and Myocardial Infarction"—Doctor James F. Hammarsten

WEDNESDAY, APRIL 20

PROBLEMS OF THE PATIENT WITH HYPERTENSION
8:30- 9:30 a.m. "Problems of Patients with Hypertension"—Doctor William Schottstaedt
9:30-10:00 a.m. Discussion
10:15-12:15 p.m. Case Presentation—Panel Discussion
12:15- 1:15 p.m. Lunch
1:15- 3:15 p.m. Special seminars for physicians*
4:00- 5:00 p.m. Film or Reference Reading

THURSDAY, APRIL 21

DETECTION AND PREVENTION OF HEART DISEASE
8:30-10:00 a.m. "Detection and Prevention of Heart Disease"—Doctor Herbert B. Hudnut, Jr.
10:15-12:15 p.m. Panel—"Problems in Prevention and Correction of Obesity"
12:15- 1:15 p.m. Lunch
1:15- 3:15 p.m. Special seminars for physicians*
4:00- 5:00 p.m. Feature—Chronic Disease Screening Unit

FRIDAY, APRIL 22

PROBLEMS OF THE PATIENT WITH RHEUMATIC HEART DISEASE
8:30- 9:30 a.m. "Problems of Patients with Rheumatic Fever"—Mrs. Theresa Morris
9:30-10:00 a.m. Discussion
10:15-12:15 p.m. Case Presentation—Panel Discussion
12:15- 1:15 p.m. Lunch
1:15- 4:00 p.m. Special seminars for physicians*
4:00- 5:00 p.m. Special Feature—"Fluorescent Antibody Technique"—Doctor L. V. Scott

SECOND WEEK

MONDAY, APRIL 25

PROBLEMS OF THE PATIENT WITH CONGENITAL HEART DISEASE
8:30- 9:30 a.m. "Problems of Patients with Congenital Heart Disease"—Doctor Galen P. Robbins
9:30-10:00 a.m. Discussion
10:15-12:15 p.m. Case Presentation—Panel Discussion
12:15- 1:15 p.m. Lunch
1:15- 2:15 p.m. Pump Oxygenator Team
2:30- 3:30 p.m. Special seminars for physicians*
4:00- 5:00 p.m. Film—Discussion

TUESDAY, APRIL 26

SPECIAL SERVICES FOR PATIENTS WITH HEART DISEASE
8:30-10:00 a.m. Panel—"The Employable Cardiac"
10:15-11:00 a.m. Discussion
11:00-12:00 noon Combined session on work simplification
12:00- 1:00 p.m. Lunch
1:00- 5:00 p.m. Special Field Trips

WEDNESDAY, APRIL 27

COMPLICATIONS OF HEART DISEASE
8:30- 9:30 a.m. "Congestive Heart Failure"—Doctor John Miller
9:30-10:00 a.m. Discussion
10:15-12:15 p.m. Case Presentation—Panel Discussion
12:15- 1:15 p.m. Lunch
1:15- 4:00 p.m. Special seminars for physicians*
4:00- 5:00 p.m. "Low Sodium Diets"—Miss Beath Heap

THURSDAY, APRIL 28

"STRIKE BACK AT STROKE"
8:30- 9:30 a.m. "Strike Back at Stroke"—Doctor Herbert Kent
9:30-10:00 a.m. Discussion
10:15-12:00 noon Case Presentation—Panel Discussion
12:00-12:30 p.m. Film, "The Challenge of Management"
12:30- 1:30 p.m. Special Low Sodium Luncheon
1:30- 2:00 p.m. Discussion of Low Sodium Luncheon
2:00- 4:00 p.m. Panel—"Psychological and Physical Rehabilitation of the Stroke Patient"
4:00- 5:00 p.m. Films, "Asphasia"—"Second Chance"

FRIDAY, APRIL 29

COMMUNITY RESPONSIBILITIES CONCERNING HEART DISEASE
8:30- 9:30 a.m. "Public Health and Heart Disease"—Doctor Kirk T. Mosley
9:30-10:00 a.m. Discussion
10:15-12:15 p.m. Discussion of four prepared cases, moderated by Doctor Kirk T. Mosley with panel
12:15- 1:15 p.m. Lunch
1:15- 3:00 p.m. Summary reports by each discipline—Recommendations for improving heart programs in Oklahoma
3:15- 4:00 p.m. Questionnaire
4:00- 4:30 p.m. Summary of the Course—Doctor Kirk T. Mosley

*SPECIAL SEMINARS FOR PHYSICIANS HEART PATIENT COURSE

April 18-29, 1960

Interested physicians may attend any part of the Heart Patient Course including the following seminars without registering for the entire course. Each session will be limited to ten physicians in addition to those enrolled in the course.

TUESDAY, APRIL 19 1960

1:15- 3:15 p.m. Room 14-E University Hospital
EARLY DETECTION AND ANTICOAGULATION THERAPY FOR CORONARY ARTERY DISEASE
Loyal Lee Conrad, M.D.

WEDNESDAY, APRIL 20, 1960

1:15- 3:15 p.m. Room 14-E University Hospital
PREVENTION, ETIOLOGICAL DIAGNOSIS, AND THERAPY OF THE HYPERTENSIVE DISEASES
William O. Smith, M.D.

THURSDAY, APRIL 21, 1960

1:15 - 3:15 p.m. Room 14-E University Hospital
METHODS AND RESULTS IN
CARDIOVASCULAR SCREENING
William Best Thompson, M.D., and
Herbert P. Hudnut, Jr., M.D.

FRIDAY, APRIL 22, 1960

1:15- 2:45 p.m. Room 14-E University Hospital
THE SIGNIFICANCE OF SYSTOLIC MURMURS
IN DETECTION OF HEART DISEASE
Robert H. Bayley, M.D.

3:00- 4:00 p.m. Room 14-E University Hospital
THE B-HEMOLYTIC STREPTOCOCCI,
THEIR BACTERIOLOGICAL AND
CLINICAL CHARACTERISTICS
Florence C. Kelly, Ph.D.

MONDAY, APRIL 25, 1960

2:30- 3:30 p.m. Room 14-E University Hospital
RESULTS OF OPEN HEART SURGERY
IN OKLAHOMA
G. Rainey Williams, M.D.

WEDNESDAY, APRIL 27, 1960

1:15- 2:45 p.m. Room 14-E University Hospital
A REVIEW OF X-RAY DIAGNOSIS IN
THE CARDIOVASCULAR DISEASES
Peter E. Russo, M.D.

3:00- 4:00 p.m. Room 14-E University Hospital
PREVENTION OF REPEATED ATTACKS OF
CONGESTIVE HEART FAILURE
John H. Miller, M.D.

Pediatric Neurology Symposium in May

A Pediatric Neurology Symposium, developed by the Division of Pediatric Neurology, Department of Pediatrics and the Office of Postgraduate Education, University of Oklahoma School of Medicine, will be held May 11, 12 and 13, 1960. This three-day meeting is a combination of the Pediatric Neurology short course which will be held May 11 in the medical center auditorium and a two-day Pediatric Neurology Symposium on May 12 and 13 in the auditorium of the Nurses Home at the medical center.

Guest speakers for the occasion will be Douglas Buchanan, M.D., Professor of Pediatrics and Neurology, University of Chicago School of Medicine, Chicago and Philip R. Dodge, M.D., Associate in Neurology, Harvard Medical School, Associate Neurologist and Associate Physician to Children's Medical Service, Massachusetts General Hospital, Boston.

Registration for the short course is $3.00 and for the symposium, $10.00. Complimentary registration will be offered interns, residents and physicians in military service.

Freedom Forum Transcription Available to County Societies

The Oklahoma County Medical Society conducted a Freedom Forum program upon which outstanding Oklahomans appeared as speakers. Because of the excellence of the presentations and the significance of the subject, the program was transcribed on long-play records which are now available through the Executive Office of the Oklahoma State Medical Association. The records could well be used for county medical society programs.

Speaking on various aspects of governmental activities were the following: Steve Stahl, Executive Vice-President of the Oklahoma Public Expenditures Council; Reverend Norman Whitehouse, Oklahoma City minister; Lewis Munn, President, Oklahoma Farm Bureau; Ferdie Deering, Editor, Farmer-Stockman; John Cantrell, Ll. B., President-Elect, Oklahoma City Downtown Rotary; and Bayard Auchincloss, Educator and Founder of "Sound Dollar, Incorporated."

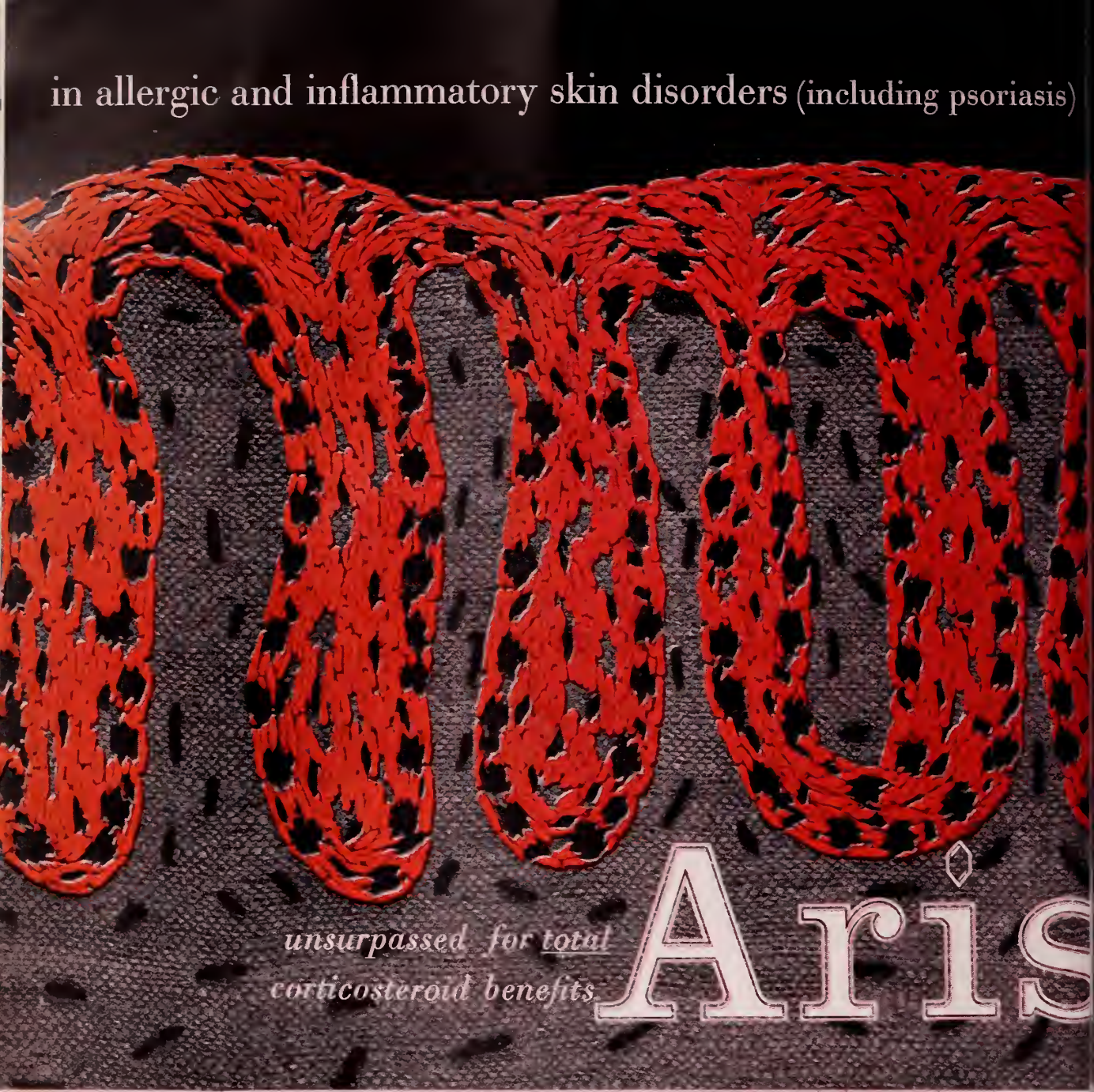
County Society Participates In Junior Stock Auction

Pottawatomie County Medical Society once again purchased the grand champion steer at the Annual Junior Livestock Show held recently in Shawnee.

Total price for the heavy Hereford steer was $441.60 or 40 cents a pound. This was a premium of 13 cents a pound over the market price of 27 cents.

A total of 69 animals were sold. Last year, the Pottawatomie Society bought every champion of the same show.

in allergic and inflammatory skin disorders (including psoriasis)

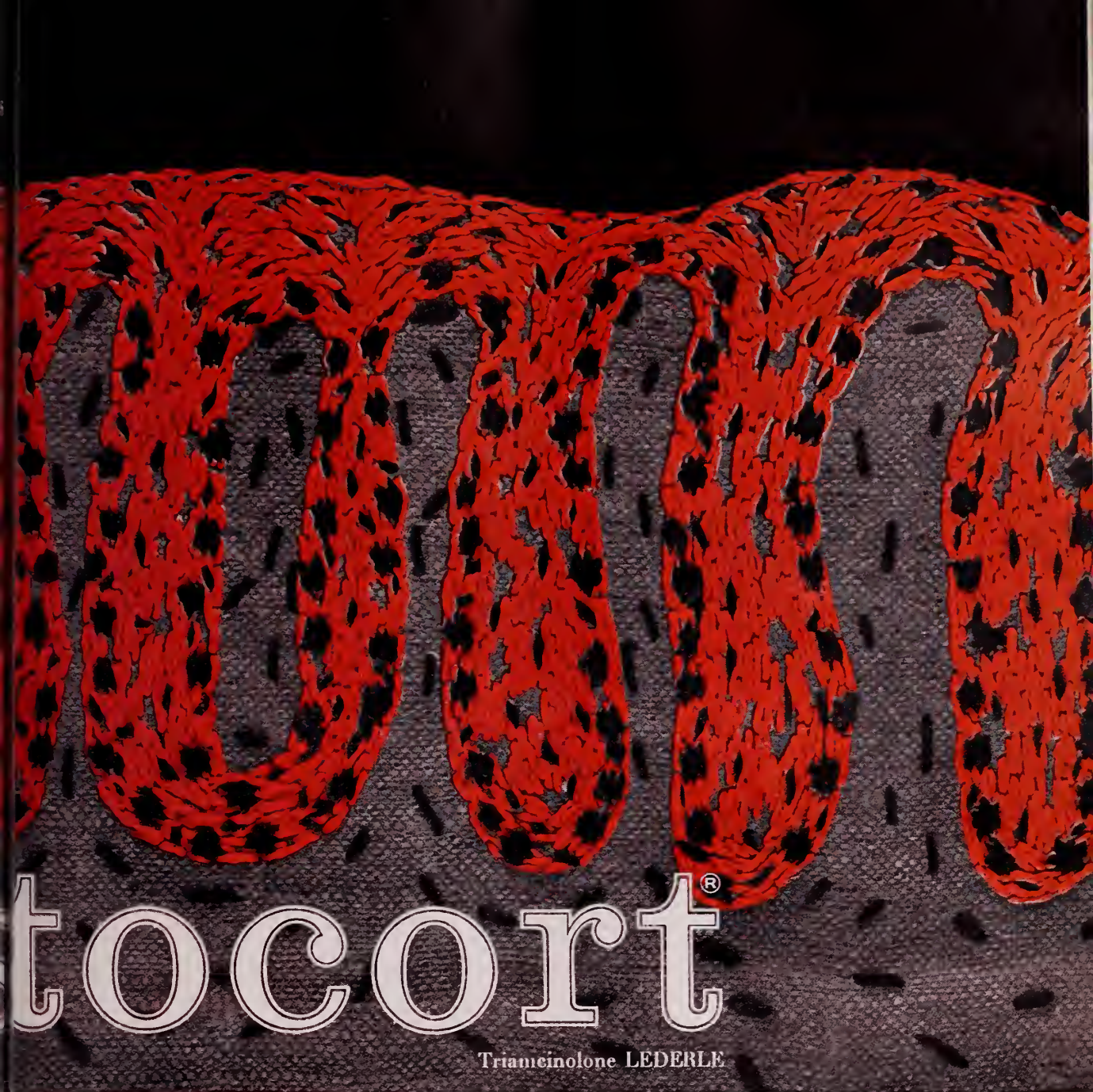


Substantiated by published reports of leading clinicians

- **effective control** of allergic and inflammatory symptoms[1-3,7,8,12-15,17,18]



- **minimal disturbance** of the patient's chemical and psychic balance[1,4-18]



At the recommended antiallergic and anti-inflammatory dosage levels, ARISTOCORT means:

- freedom from salt and water retention
- virtual freedom from potassium depletion
- negligible calcium depletion
- euphoria and depression rare
- no voracious appetite — no excessive weight gain
- low incidence of peptic ulcer
- low incidence of osteoporosis with compression fracture

Precautions: With ARISTOCORT all traditional precautions to corticosteroid therapy should be observed. Dosage should always be carefully adjusted to the smallest amount which will suppress symptoms.

After patients have been on steroids for prolonged periods, discontinuance must be carried out gradually over a period of as much as several weeks.

Supplied: 1 mg. scored tablets (yellow); 2 mg. scored tablets (pink); 4 mg. scored tablets (white); 16 mg. scored tablets (white).

Diacetate Parenteral (for intra-articular and intrasynovial injection). Vials of 5 cc. (25 mg./cc.).

References: 1. Feinberg, S. M.; Feinberg, A. R., and Fisherman E. W.: *J.A.M.A.* 167:58 (May 3) 1958. 2. Epstein, J. I., and Sher wood, H.: *Conn. Med.* 22:822 (Dec.) 1958. 3. Friedlaender, S., an Friedlaender, A. S.: *Antibiotic Med. & Clin. Ther.* 5:315 (May 1958. 4. Segal, M. S., and Duvenci, J.: *Bull. Tufts N.E. Medica Center* 4:71 (April-June) 1958. 5. Segal, M. S.: Report to th A.M.A. Council on Drugs, *J.A.M.A.* 169:1063 (March 7) 1958 6. Hartung, E. F.: *J. Florida Acad. Gen. Practice* 8:18, 1957 7. Rein, C. R.; Fleischwager, R., and Rosenthal, A. L.: *J.A.M.A* 165: 1821 (Dec. 7) 1957. 8. McGavack, T. H.: *Clin. Med.* (June 1959. 9. Freyberg, R. H.; Berntsen, C. A., and Hellman, L. *Arthritis & Rheumatism* 1:215 (June) 1958. 10. Hartung, E. F. *J.A.M.A.* 167:973 (June 21) 1958. 11. Zuckner, J.; Ramsey, R. H. Caciolo, C., and Gantner, G. E.: *Ann. Rheumat. Dis.* 17:398 (Dec. 1958. 12. Appel, B.; Tye, M. J., and Leibsohn, E.: *Antibiotic Med & Clin. Ther.* 5:716 (Dec.) 1958. 13. Kalz, F.: *Canad M.A.J.* 79:400 (Sept.) 1958. 14. Mullins, J. F., and Wilson, C. J.: *Texas J Med.* 54:648 (Sept.) 1958. 15. Shelley, W. B.; Harun, J. S. and Pillsbury, D. M.: *J.A.M.A.* 167:959 (June 21) 1958 16. DuP is E. L.: *J.A.M.A.* 167:1590 (July 26) 1958. 17. McGavack, T. H. Kao, K. T.; Leake, D. A.; Bauer, H. G., and Berger, H. E.: *Am J. M. Sc.* 236:720 (Dec.) 1958. 18. Council on Drugs: *J.A.M.A* 169:257 (January) 1959.

Lederle LEDERLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, N. Y

Preceptors Listed for Medical School

Three new preceptors—E. C. Lindley, M.D., of Duncan; Robert W. Lowrey, M.D., of Poteau, and J. A. Graham, M.D., of Pauls Valley—have been appointed to the faculty of the University of Oklahoma School of Medicine. Doctor Graham formerly served as an associate preceptor.

Preceptors re-appointed for 1960-61 were: Lynn C. Barnes, M.D., Nowata; Edward T. Cook, Jr., M.D., Anadarko; Walter H. Dersch, Jr., M.D., Shattuck; Joe L. Duer, M.D., Woodward; George Gathers, M.D., Stillwater; Robert B. Gibson, M.D., Ponca City; Burdge F. Green, M.D., Stilwell;

C. K. Holland, M.D., McAlester; L. A. S. Johnston, M.D., Holdenville; W. C. McCurdy, M.D., Purcell; E. A. McGrew, M.D., Beaver; Malcolm Mollison, M.D., Altus; Cody Ray, M.D., Pawhuska; Edward T. Shirley, M.D., Wynnewood; Carlton E. Smith, M.D., Henryetta; George A. Tallant, M.D., Frederick, and Tom L. Wainwright, M.D., Mangum.

Medical Assistants to Hold Seminar

The Oklahoma State Medical Assistants Society has announced the second annual Seminar for Medical Assistants to be held on the Oklahoma State University campus, Stillwater, June 11-12, 1960.

The purpose of this workshop is to give medical assistants additional training and to broaden their understanding to enable them to do a more effective job for the physicians and their patients. The program is aimed specifically at those medical assistants who have an interest in education and self-improvement as well as better standards of training. Background information of value to the medical assistant will be stressed together with practical advice for meeting day-to-day problems.

The faculty will be comprised of well know members of the medical profession, educational leaders of the University and other professional experts in a variety of fields. Topics to be covered include: "Psychology of Human Behavior," "Basic Economics," "Personal Appearance and the Office," "Collection Problems," "Office Systems," "Problems of Prepayment of Medical Costs," and "Professional Liability Problems of Medical Assistants." Registration fee will be $15.00.

The Society extends a most cordial invitation to its membership and to other interested medical assistants. Any medical assistant or others employed in a physician's office are eligible to attend this Seminar.

Steve Stahl Honored

Executive Vice-President of the Oklahoma Public Expenditures Council and Chairman of the National Taxpayer's Conference, Steve Stahl, was honored February 22, 1960, with the presentation of a gold medal and citation by the Freedoms Foundation at Valley Forge. The citation read:

> "The Trustees, Directors and Officers of Freedoms Foundation at Valley Forge announce with pleasure the selection of Steve Stahl by the distinguished National & School Awards Jury to receive a George Washington Honor Medal Award for Television Series on Economic Education, "The Voice of the Taxpayer." An outstanding achievement in helping to bring about a better understanding of the American Way of Life during 1959."

Late last year, in a joint endeavor of the Oklahoma Public Expenditures Council and the Oklahoma State Medical Association, Mr. Stahl directed a special, one-hour program of the "Voice of the Taxpayer" series on KOCO-TV. The program, "Old Age and You," was presented by Louis M. Orr, M.D., President of the American Medical Association; Rita Campbell, Ph.D., Consultant Economist to the American Enterprise Association, Washington, D.C.; N. D. Helland, Executive Director, Oklahoma Blue Cross-Blue Shield Plans; Tom Sorey, Oklahoma City architect and Board member of Senior Citizens, Incorporated; and Mr. Stahl.

Rocky Mountain Cancer Conference Will Be in July

The 14th annual Rocky Mountain Cancer Conference will be held in the new Denver Hilton Hotel in Denver, Colorado on July 20-21, 1960. Nearly 900 physicians from all over the nation are expected to attend the two-day scientific session, which is approved for 10 AAGP Category I Credits.

The regional cancer-control meeting is jointly sponsored each year by the Colorado division of the American Cancer Society and the Colorado State Medical Society.

Tentative program plans call for a symposium on "Skin Cancer" on Wednesday morning, July 20 and a symposium on "Thyroid Lumps" on the following morning. Afternoon sessions on both days will be devoted to papers on cancer detection and treatment by six outstanding physicians.

Symposium participants and speakers are: R. Lee Clark, Jr., M.D., Houston, Texas; A. James French, M.D., Ann Arbor, Michigan; Roy L. Kile, M.D., Cincinnati, Ohio; Wendell G. Scott, M.D., St. Louis, Missouri; H. W. Schmidt, M.D., Rochester, Minnesota; and Willard P. VanderLaan, M.D., La Jolla, California.

E. Vincent Askey, M.D., of Los Angeles, California, President-Elect of the American Medical Association and Warren H. Cole, M.D., of Chicago, Illinois, President of the American Cancer Society, will also participate in the conference.

Site of the Cancer Conference will be the new Denver Hilton Hotel which is completely air-conditioned.

Alpha Omega Alpha Spring Lecture

Robert H. Ebert, M.D., Professor of Medicine, Western Reserve University School of Medicine, Cleveland, Ohio, will present the Annual Alpha Omega Alpha Spring Lecture to be held on April 19, at 4:00 p.m., in the auditorium of the University of Oklahoma School of Medicine.

Colorado to Host PG Course in Pediatrics

The Department of Pediatrics and the Office of Postgraduate Medical Education, University of Colorado School of Medicine will present a Course in Pediatrics, September 1-6, 1960 at the Stanley Hotel, Estes Park, Colorado.

This five-day postgraduate course, designed for the practicing pediatrician, will be presented in two parts. Physicians may register for the entire course or for either part. Part 1, September 1-3, will be devoted to Clinical and Research Advances in Pediatrics. Child Guidance Problems will be discussed during the second portion of the meeting on September 5-6.

A great variety of subjects will be covered and seminars on recent advances in various pediatric subspecialties will be held as part of the program.

Tuition fee will be $60.00 for the entire course, or $30.00 for either of the two parts.

Regularly scheduled bus service will be available between Estes Park and Denver.

Detailed information is available from the Office of Postgraduate Medical Education, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver 20, Colorado.

Physicians Art Work To Be Exhibited

The 23rd annual exhibition of art works by American physicians will be held June 13 through June 18, 1960, at the Miami Beach Exhibition Hall and Auditorium, it was announced by Lewis M. Johnson, M.D., President of the American Physicians Art Association.

Held in conjunction with the annual convention of the American Medical Association, the show will include over 300 works of art in oil, water color, sculpture, crafts, photography and lithography.

Participants and prospective exhibitors may obtain further information from Kurt F. Falkson, M.D., 7 East 78th Street, New York City, Secretary of the American Physicians Art Association.

Five More Courses Offered by A.C.P.

Five courses remain on the postgraduate schedule of the American College of Physicians. Two earlier courses were held on "Recent Advances in Pharmacotherapy" in Seattle, Washington and "Current Concepts in Gastroenterology" in New Orleans, Louisiana.

"Dermatology for the Internist," course number three, will be presented at the Medical Center, University of Michigan, Ann Arbor, April 25-29, 1960. Designed especially for internists, although some of the common dermatoses will be discussed, major emphasis will be placed on those diseases which fall in the province of both the dermatologist and internist.

The University of Pennsylvania School of Medicine and the Department of Public Health and Preventive Medicine, Philadelphia, will host course number four, "Early Detection and Prevention of Disease," May 9-13, 1960. This course will give a critical evaluation of the possibilities and techniques for the early diagnosis of disease and the prevention of disease where prevention is possible.

The National Heart Institute, Bethesda, Maryland, will present the fifth course on "Current Research in Cardiovascular Disease," May 16-20, 1960. Here, the internists will hear a discussion of many of the facets of cardiovascular disease which are under investigation at the present time. The research program of the Heart Institute will serve as a basis for the presentations, but no attempt will be made to cover all of the basic studies nor to cover the entire field of cardiovascular disease.

The sixth course, "The Hypertensive Diseases: Diagnostic and Therapeutic Procedure in Essential, Adrenal and Renal Hypertension" will be presented by the Massachusetts Memorial Hospitals in Boston, May 23-26, 1960. Considerations of problems of hypertension from the moment of its recognition in a patient by a physician, through its proper clinical classification made with the help of various diagnostic and prognostic procedures, and the selection of therapy including long term management by all available means, both medical and surgical will be made.

Final presentation will be "Internal Medicine" at the Indiana University School of Medicine, Indianapolis, June 20-24, 1960. Participants will deal with selected subjects in the field of internal medicine and will emphasize pathological physiology and mechanisms of disease.

Registration Fees

The College, believing that its Postgraduate Course Program should be essentially self-supporting, announces new matriculation fees—$60.00 per week for members of the College; $80.00 per week for non-members.

All registrations must be made through the Executive Offices of the College, 4200 Pine Street, Philadelphia 4, Pennsylvania.

Deaths

HARRY PAUL KEMMERLY, M.D.
1901-1960

Harry Paul Kemmerly, M.D., Tulsa anesthesiologist, died February 29, 1960.

Born in Shattuck, Oklahoma in 1901, Doctor Kemmerly was a teacher and later served as principal of a grade school in Hominy.

After graduating from the University of Arkansas School of Medicine in 1928, he established his practice in Tulsa.

WILLIAM C. MILLER, M.D.
1890-1960

William C. Miller, M.D., retired Guthrie physician, died February 29, 1960 in Tahlequah.

A native of Galesburg, Illinois, Doctor Miller graduated from George Washington University in 1917. He practiced in Guthrie for 35 years before retiring in 1956.

In 1957, Doctor Miller was honored when the Oklahoma State Medical Association presented him with a Life Membership in recognition of his years of service to the profession.

Coming Meetings

UNIVERSITY OF OKLAHOMA MEDICAL CENTER
POSTGRADUATE PROGRAM*
Individual Postgraduate Courses

ORTHOPEDIC SYMPOSIUM—April 22 and 23.
ATHLETIC INJURIES

OKLAHOMA ASSSOCIATION OF HOUSE STAFF PHYSICIANS—May 6.

PEDIATRIC NEUROLOGY—May 12 and 13. This symposium to be held in conjunction with the Wednesday Short Course on May 11.

CARCINOMA OF THE SKIN—May 27-28. Surgery, Radiology, Pathology Conference.

SERIAL POSTGRADUATE COURSE
Postgraduate Division
Oklahoma City, Oklahoma
1959-1960

Wednesday Short Courses
3:30 to 8:30 p.m.

May 11	Neurological Diseases in Childhood
June 8	Practical Considerations of Neurological Problems

*Further information may be obtained from the Office of Postgraduate Education, University of Oklahoma School of Medicine, 801 N.E. 13th Street, Oklahoma City, Oklahoma.

24th ANNUAL MEETING
OKLAHOMA SOCIETY OF MEDICAL TECHNOLOGISTS

April 22-24, 1960 **Lake Texoma Lodge**
Kingston, Oklahoma

The 24th Annual Meeting of the Oklahoma Society of Medical Technologists will be held at Lake Texoma Lodge, Kingston, Oklahoma, April 22-24, 1960. Eight guest speakers are listed on the program. Registrations should be addressed to Miss Mary Ann McClure, M.T. (ASCP), Registration Chairman, Valley View Hospital, Ada, Oklahoma.

METHODIST LECTURESHIP IN MEDICINE

May 7, 1960 **Tom S. Lubbock High School**
Lubbock, Texas

The Methodist Hospital Lectureship in Medicine will be held Saturday, May 7, 1960 in the Tom S. Lubbock High School, Lubbock, Texas. Guest lecturers will be William Dameshek, M.D., Boston, Massachusetts; William H. Crosby, M.D., Washington, D.C.; and Joseph M. Hill, M.D., Dallas, Texas. Inquiries may be directed to Patrick H. Pappas, M.D., 3621 21st Street, Lubbock, Texas.

POSTGRADUATE COURSE IN PEDIATRICS

The University of Colorado School of Medicine

September 1-6, 1960 **The Stanley Hotel**
Estes Park, Colorado

The Department of Pediatrics and the Office of Postgraduate Medical Education, University of Colorado School of Medicine will present a Course in Pediatrics at the Stanley Hotel, Estes Park, Colorado, September 1-6, 1960. Further information may be found in the News Section of this issue of **The Journal.**

ROCKY MOUNTAIN CANCER CONFERENCE

July 20-21, 1960 **Denver Hilton Hotel**
Denver, Colorado

The 14th Annual Rocky Mountain Cancer Conference will be held in Denver, July 20-21, 1960 with headquarters at the Denver Hilton Hotel. Inquiries should be directed to Rocky Mountain Cancer Conference, 835 Republic Building, Denver 2, Colorado.

SECOND ANNUAL
OREGON CANCER CONFERENCE

July 7-8, 1960 **Portland, Oregon**

The second Annual Oregon Cancer Conference will be held in Portland, July 7-8, 1960. A copy of the complete program may be obtained by writing to Roscoe K. Miller, Executive Secretary, Oregon State Medical Society, 2164 S. W. Park Place, Portland 5, Oregon.

6th INTERNATIONAL CONGRESS OF INTERNAL MEDICINE

August 24-27, 1960 **Basle, Switzerland**

The 6th International Congress of Internal Medicine will convene in Basle, Switzerland, August 24-27, 1960. Simultaneous translation for the principal speakers will be given in German, French and English. Programs and registration forms are available from the 6th International Congress of Internal Medicine, Steinentorstrasse 13, Basle 10, Switzerland.

25 YEARS AGO



Articles published in *The Journal* of the Oklahoma State Medical Association, April, 1935.

WHAT THE PUBLIC THINKS OF DOCTORS

Rev. Sidney H. Babcock, D.D.
Holdenville

The public mind is sensitive. Every act of every profession reacts upon it. Moreover, the public mind is vacillating. What it thinks today, it may, or may not, think tomorrow, its judgments today may be overturned by the facts of tomorrow. Popular opinions, sympathetic or unsympathetic, are not always true opinions. The public mind changes with the shifting scenes.

Nevertheless, the public mind is positive and persistent. There are positive opinions which carry through the years and certain public judgments which persist from one generation to another. Whether you agree with me, or not, that the soul persists after death, you will probably agree with me that there are certain positive judgments of the public mind that persist from one generation to another.

The public persists in giving to your profession a very high rank, perhaps the very highest rank. It places the art of healing and the art of disease prevention among the noblest of all arts. This it has done despite the fact that here and there on the horizon there have appeared quasi-scientific and quasi-religious cults that have sought to discount the medical profession.

The public gives to doctors the high honor of guardians of the public health. In this the doctor has a dual relation. He must make his livelihood by attacking and destroying the causes of the very diseases which call for his services which in turn provide his livelihood. The Great Physician said one time, "A house divided against itself shall fall, " but He also said, "Whoever shall lose his life shall preserve it." The great fight doctors have made, and are constantly making, to conquer disease has greatly endeared them in the public mind.

The fine way in which our modern doctors spare neither time nor expense to bring to our communities the best medical thought, the best preventative measures, and, when necessary, the best curative and surgical skill fully justifies the public mind in giving to them the high place in our society that it certainly does.

Then, at last, when the twilight shadows gather and we catch glimpses of gold on the other shore, the doctor still watchful, still faithful, becomes more than a skilled scientist, more than an honored servant of the community, more than a valiant guardian of the public health—now he is "a friend that sticketh closer than a brother."

Have You Heard?

GERALD ROGERS, M.D., Oklahoma City, spoke before the Birmingham Obstetrical and Gynecology Society, in Birmingham, Alabama recently. Subject for his speech was, "The Treatment of Postoperative Vesicovaginal Fistulas."

PAUL B. LINGENFELTER, M.D., Clinton, was recently elected to serve as president of the Clinton Chamber of Commerce. He has served three years as a director of the organization.

R. D. HAYS, M.D., has opened offices in the City Hall building in Pawnee. Doctor Hays, a graduate of the University of Tennessee College of Medicine, has been taking special training at Hillcrest Medical Center since his internship there.

ROBERT MEYER, M.D., recently joined the staff of the Bethany Medical Center at 205 N.W. Main in Bethany. A graduate of the University of Minnesota School of Medicine, Doctor Meyer has practiced in Minneapolis for the past nine years.

R. M. SHEPARD, SR., M.D., Tulsa chest physician, was recently chosen "Doctor of the Year" by the Woman's Auxiliary to the Tulsa County Medical Society. Doctor Shepard's career has included terms as President for the American Trudeau Society and of the Tulsa County Medical Society. He has been a director of the Tulsa County Health Association since 1930, and was a national director of the tuberculosis association for 27 years.

PHYSICIAN PLACEMENT

Dermatology

Gordon Harold Ekblad, M.D., Field Medical Service School, Camp Pendleton, California, 53, married, University of Minnesota, 1930, available January 1960.

Lealand L. Clark, M.D., 200 1st St., S.W., Rochester, Minnesota; age 28; married; Columbia University College Physicians and Surgeons 1956; Board eligible July 1960; Fellows' Assoc., Mayo Foundation; Military service will be completed Sept. 1962; desires group or associate practice; available July 1960.

Joseph William Pidgeon, M.D., 5333 Littlebow Road, Rolling Hills, California; age 41; married; George Washington University 1951; Board eligible June 1960; Active Air Force Reserves; available July 1960.

General Practice

Johnny Bill Delashaw, U.S.P.H.S. Hospital, Wyman Park Drive and 31st Street, Baltimore 11, Maryland, age 27, married, University of Texas Medical Branch, 1959, available July 1960.

Robert Goodman, M.D., 63 Old Gate Lane, Powers Lake, North Dakota; age 56; married; University of Manitoba, 1930; no military obligations; available anytime 1960.

Richard Fred Harper, M.D., Conway Memorial Hospital, Monroe, Louisiana; age 28, married; Oklahoma University, 1956; available July 1960; prefer associate practice.

Sherman Allen Hope, M.D., 1105 Yale Ave., Panama City, Florida, PO 3-6656, age 27, married, University of Oklahoma School of Medicine, 1957, available July 1960.

Internal Medicine

Richard I. Hochman, M.D., 37, Badger Road, Annapolis, Maryland, 31, married, New York University, 1952, available October 1, 1960.

Edward John Hertko, M.D., 2117 S. 3rd Ave., Maywood, Illinois; age 30; married; University of Illinois, 1954; no military obligations; available October 1960.

Joseph Chester Maternowski, M.D., 319 Highlake, Ann Arbor, Michigan; age 35; married; St. Louis University, 1953; eligible board of Internal Medicine & Allergy; veteran; specialty, Internal Medicine & Allergy; available July 1, 1960.

Van B. Saye, Jr., M.D., 1230 Brockenbraugh Court, Metairie, Louisiana, age 28, married, Medical College of Georgia, 1954, Air Force Reserve, available July 1, 1960.

Locum Tenens

Harold W. Calhoon, M.D., Wesley Hospital, Oklahoma City. One year residency in Internal Medicine. Available July 1 to September 15th, 1960.

Arnold Giesbrecht, M.D., Hallock, Minnesota, age 34, married, graduate Winnipeg, Manitoba, 1957, prefers suburban or locality close to cities.

James D. Green, M.D., second year resident in Internal Medicine at St. John's Hospital, Tulsa, desires two weeks' tenure between now and July 1. Write to St. John's or call RIverside 3-3059.

Charles L. Maimbourg, M.D., Campus Apartments No. 5, Elf Street, Durham, North Carolina; Oklahoma University School of Medicine 1958; desires General Practice and/or Anesthesia during first two or three weeks of July 1960.

Russell M. Preston, M.D., 476 San Juan, Oak Harbor, Washington, desires six month's work in General Practice between time of separation from military service to beginning of residency training, January-July, 1960.

Obstetrics and Gynecology

Lynn W. Abshere, M.D., 708 N.E. 14th, Oklahoma City, age 31, married, University of Oklahoma School of Medicine, 1956, American College of OB-GYN, no military obligations, available July 1, 1960.

Edmond Michael Brophy, M.D., 1823 Portsmouth, Westchester Illinois; married; University of Illinois, 1946; Board eligible upon completion of practice requirements; no military obligations; available July, 1960.

William Edgar Carlisle, M.D., 634 E. Patton Ave., Montgomery, Alabama; age 29; married; Tulane University School of Medicine 1953; Jr. Fellow American College of Ob-Gyn; interested in associate or group practice; available July 1960.

W. Paul Dickinson, M.D., 554 S. Summit, Ft. Worth, Texas; age 37; married; University of Oklahoma School of Medicine, 1948; board eligible July 1960; available August 1960.

Robert Russell Fahringer, M.D., P.O. Box 148, Danville, Pennsylvania; age 35; married; Jefferson Medical College of Philadelphia, 1953; inactive military status, USNR; prefers full or part-time teaching; available February 1960.

Joseph William Herbert, M.D., 2701 N. Azelea St., Victoria, Texas; age 40; married; Southwestern Medical School, 1945; Fellow American College of Ob-Gyn; desires associate or solo practice; no military obligations; available March 1960.

David H. Holmes, M.D., 1017 Grovena Drive, St. Louis, Missouri, 37, married, Washington University, St. Louis, Veteran, available July 1, 1960.

Gerald R. Keilson, M.D., Medical Arts Building, Dallas, Texas, age 31, married, University of Texas, 1953, board qualified, veteran, available in July 1960.

Nejdat Mulla, M.D., 1110 Belmont Avenue, Youngstown, Ohio, age 36, married, University of Geneva, Switzerland, 1952, American College of OB-GYN, not eligible military service, available July 1, 1960.

Harold G. Ray, M.D., 212 Darien Place, Box 503, Balboa Heights, Canal Zone, age 28, married, University Arkansas School of Medicine, 1955, now in active reserve, available July 1, 1960.

Harold E. Reid, M.D., 519 N. Lincoln St., Stanton, Michigan; age 42; married; University of Michigan, 1942; OB-GYN, also interested in industrial; available February 1960.

Juan C. Ruiz, M.D., 350 Park Ave., Lexington, Kentucky; age 36; married; University of San Marcos Medical School 1952; no military obligations; available July 1, 1960.

Alvon C. Winegar, M.D., 1940 Michigan, Benton Harbor, Michigan, age 41, married, Wayne University, 1942, no military obligation, available early 1960.

Pediatrics

H. M. McClintock, M.D., 5712 St. John St., Kansas City, Missouri, 29, married, Baylor University, 1955, veteran, available December 1960.

Ophthalmology

Howard A. Dinsdale, M.D., 4624 Briarfield Road, Columbia, South Carolina; age 29; married; University of Nebraska 1954; Board Certified; available October 11, 1960.

Ira H. Kaufman, 75 S. Middle Neck Road, Great Neck, New York; age 31; married; Cornell University 1953; Board certified; desires group or associate practice; available summer 1960.

Orthopedics

Newsom Stool, M.D., c/o May Clinic, Rochester, Minnesota; age 30; married; Baylor University 1953; Board eligible; available July 1960.

Surgery

(Name on Request) 32 years old, married, Tulane, 1952, veteran, board eligible.

Clarence I. Britt, M.D., 1650 Neil Avenue, Apt 15, Columbus, Ohio; age 32; married; Ohio University College of Medicine, 1951; American Board of Surgery 1959; eligible American Board of Thoracic Surgery, 1960; inactive reserve; available July 1960.

Delbert H. McGinnis, M.D., Box 75, Clinton-Sherman AFB, Oklahoma; age 28; married; end military obligation July 3, 1960; interested in general practice; available July 4, 1960.

Duane M. Clement, M.D., 310 Bates, Boulder, Colorado; age 39; married; University of Michigan 1944; Board certified; member F. A. Coller Surgical Society; desires group or associate practice; available April 1960.

Alice F. Gambill, M.D., 1601 Clover Lane, Fort Worth, Texas; age 36; married; Oklahoma University School of Medicine 1949; member AAGP; desires general or group practice; available May 1960.

Donal Wray Steph, M.D., 8519 Craighill, Dallas, Texas; age 30; married; Southwestern Medical School 1953; board eligible July 1960; available July 1960.

Clinton K. Higgins, M.D., 215 Bacon Ave., Naval Base, Norfolk, Virginia; age 62; married; Washington University 1927; Board Certified; desires administration, college health physician, group association; available May 1960.

Urology

Alex Grossman, M.D., V.A. Center, Temple, Texas, 42, married, University of Texas, 1951, board certified, veteran, available now.

John A. Malley, M.D., 3404 Sheffield Ave., Los Angeles, California; age 32; married; Marquette University 1955; Board certified; no military obligations; available July 1, 1960.

MISCELLANEOUS ADVERTISEMENTS

FOR SALE: Autoclave, Castle, electric. Originally purchased for $1866.75 in 1956. Contact Walt Allen, 116 North Fourth Street, Chicaksha, Oklahoma.

FOR SALE or RENT: Office building, equipment and supplies, including x-ray. Residence also available. Contact Box F, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: $40,000 per year General Practice. 14 room completely equipped, air conditioned clinic. County seat town of 4,000 and 20,000 trade territory with new 40 bed hospital. Small down payment and terms to suit buyer. Write Key J, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: General practice, established six years, in oil and ranching community. Town population is 3,000 with trade area over 15,000. Closest physician 30 miles. Netting $20,000 to $24,000 per year. Located in modern, leased clinic building. Presently in partnership but continued partnership not mandatory. 18-bed, modern, community hospital. Only two physicians in town. Reason for selling, physician returning to Hawaii. No down payment necessary. Contact William G. Mays, M.D., Fairfax, Oklahoma.

WANTED: Young General Practitioner, without military obligations, to become partner in established clinic and hospital practice. $1000.00 per month to start. Contact Neumann-Ottis Clinic, Okarche, Oklahoma.

DEPUTY FLIGHT SURGEON wanted for Federal Aviation Agency. The 40 hours-per-week position pays an annual salary of $12,555, plus Civil Service benefits including a retirement program, annual and sick leave policy, group life insurance and others. Physician must have completed the basic course in Aviation Medicine prescribed by the U.S. Military Service, and have three years professional experience in internal medicine. Write FAA, Region 2, Box 1689, Ft. Worth 1, Texas.

WANTED: Internist to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR RENT: Office space for two doctors, modern, air conditioned building. 3832 N. May Ave., Oklahoma City. Complete maintenance. Free parking space for patients. WI 2-5531.

WANTED: Surgeon to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: Practice, office building and equipment in Harlingen, Texas. 1,000 square feet of floor space, six rooms equipped for Eye, Ear, Nose and Throat work, small laboratory and dark room. Building valued at $15,821.00. Equipment $6,720. Contact Edward M. Loyd, M.D., P.O. Box 2061, Harlingen, Texas.

FOR SALE: General practice partnerships established 7 years in prosperous oil and agriculture community. Town of 5000 and trade area over 15,000 population. Gross receipts exceed $50,000 annually. New clinical building for sale or rent. Practice is priced reasonably and terms can be arranged. Owners going into residency. Contact Box L, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: New $3250.00 Profexray, 100 MA, 100 KV X-ray unit with: rotating anode tube and examining type table with built in recipromatic bucky and table mounted tube stand, foam rubber pad for table and heel stirrups, transformer and control with shock proof cables including 1/30th second timer, one floor mounted cassette holder, one Videx rectangular cone with center light. Price $2500.00. Can be seen at Merkle X-ray Co., Tulsa, Oklahoma. Contact E. Malcolm Stokes, M.D., 2021 South Lewis, Tulsa.

NEGRO PHYSICIAN wanted for Chickasha, Oklahoma. Contact R. C. Alexander, 917 South 2nd Street, Chickasha, Oklahoma.

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lights the road to recovery in 80 per cent of cases

In the treatment of depression Tofrānil has established the remarkable record of producing remission or improvement in approximately 80 per cent of cases.[1-7]

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a potent thymoleptic . . .
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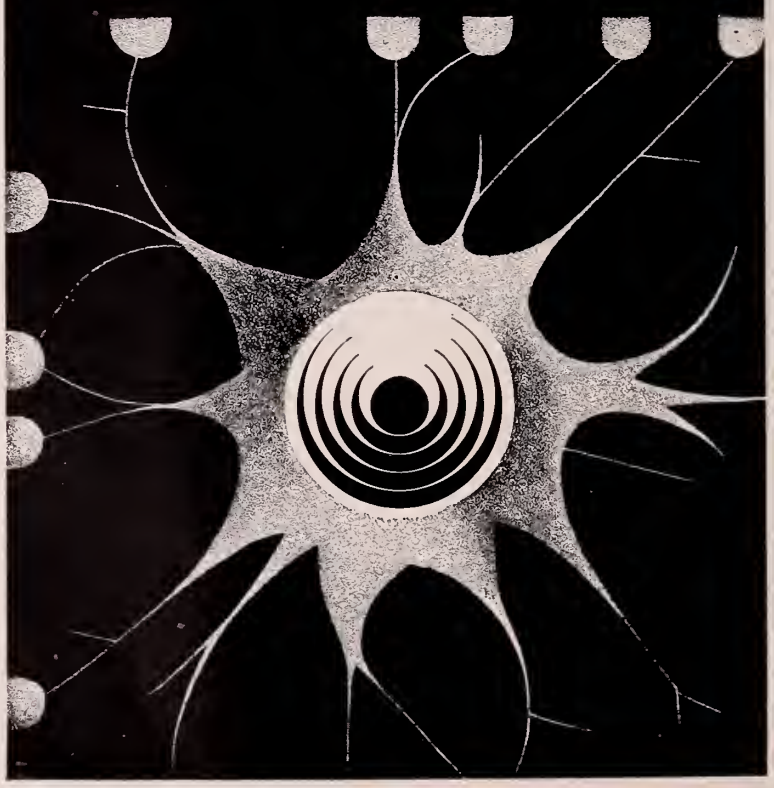
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References: 1. Ayd, F. J., Jr.: Bull. School Med., Univ. Maryland *44*:29, 1959. 2. Azima, H., and Vispo, R. H.: A.M.A. Arch. Neurol. & Psychiat. *81*:658, 1959. 3. Lehmann, H. E.; Cahn, C. H., and de Verteuil, R. L.: Canad. Psychiat. A. J. *3*:155, 1958. 4. Mann, A. M. and MacPherson, A. S.: Canad. Psychiat. A. J. *4*:38, 1959. 5. Sloane, R. B.; Habib, A., and Batt, U. E.: Canad. M.A.J. *80*:540, 1959. 6. Straker, M.: Canad. M.A.J. *80*:546, 1959. 7. Strauss, H.: New York J. Med. *59*:2906, 1959.

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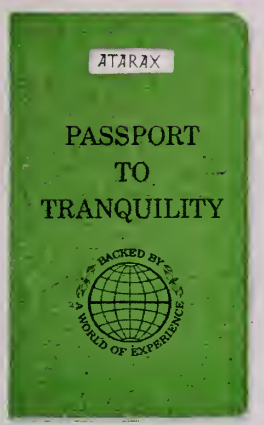
Winkelstein, A.: Am. J. Gastroenterol. *32*:66-70 (July) 1959.

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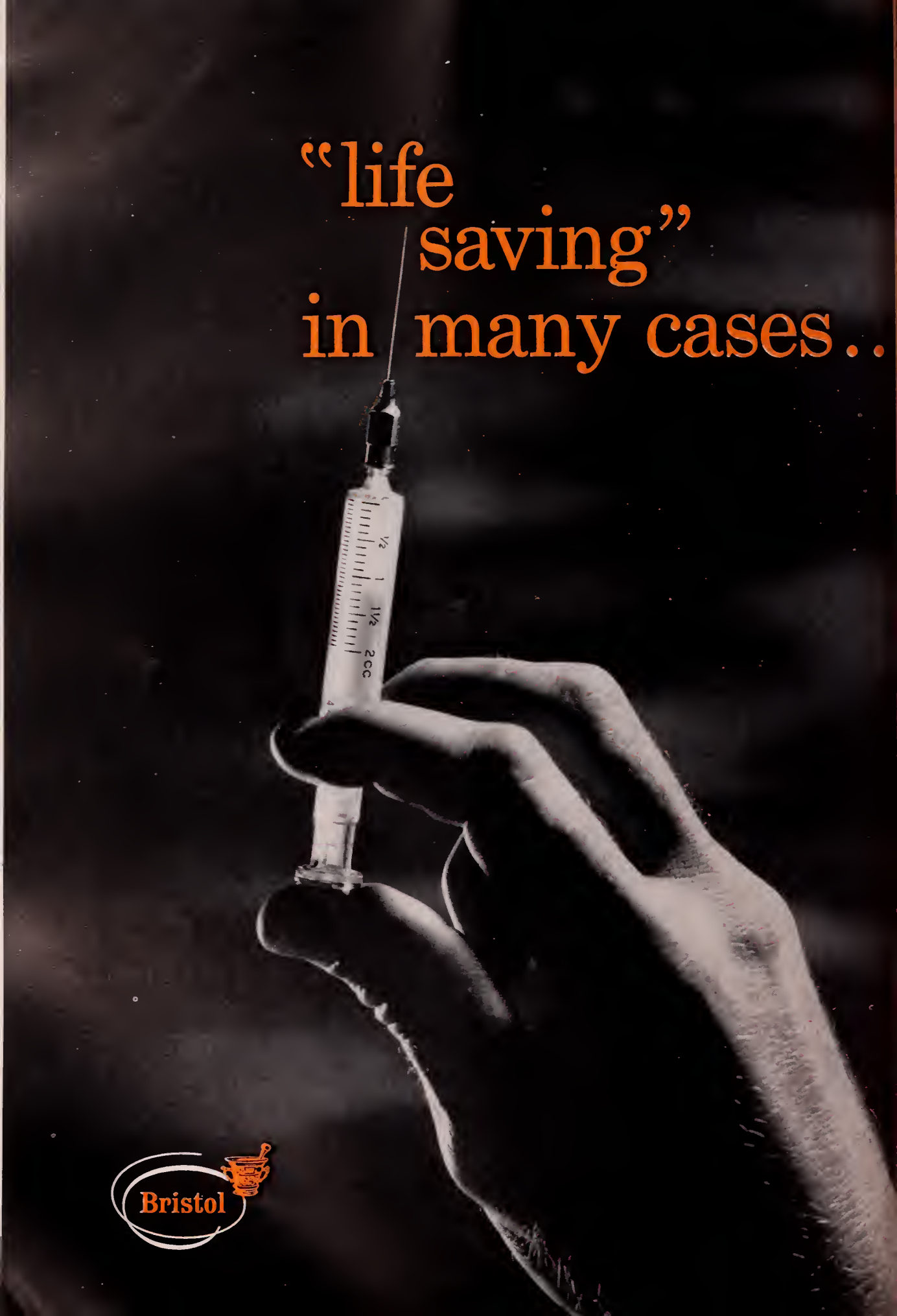
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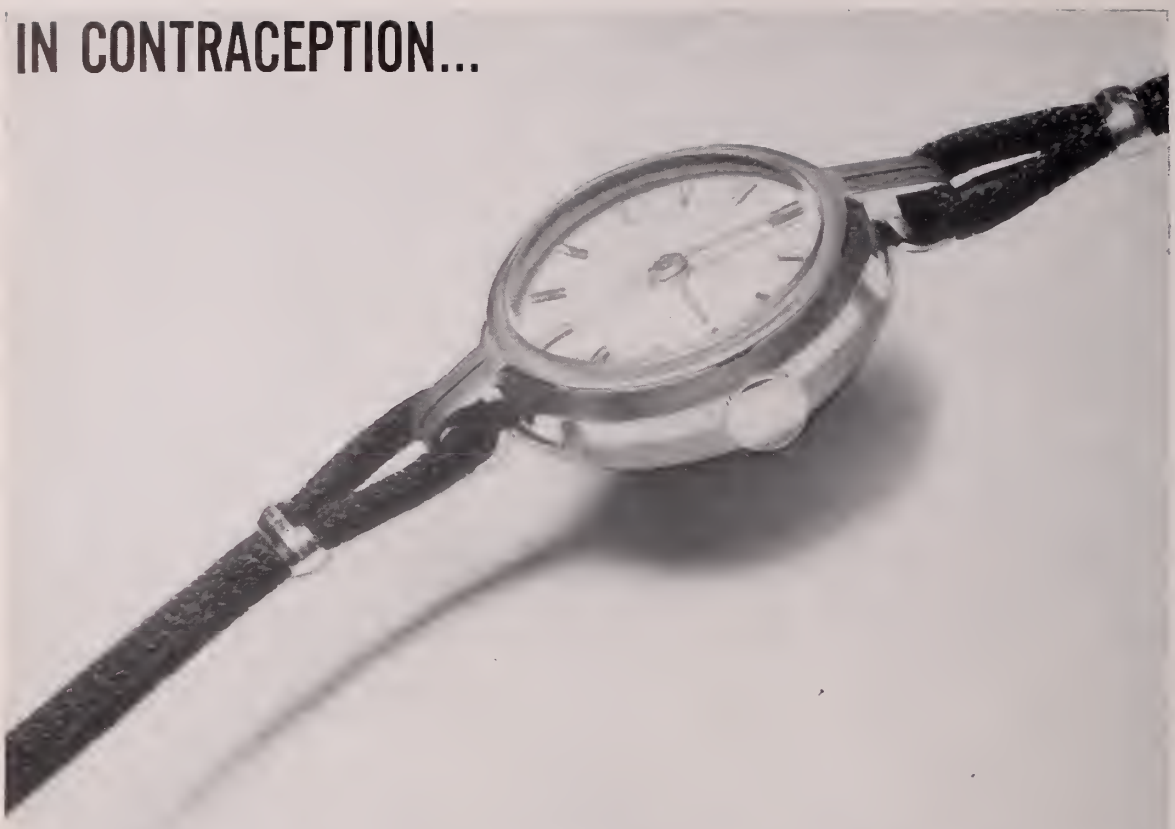
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REFERENCES: 1. Yow, E. M.: Practitioner 182:759, 1959. 2. Yow, M. D., and Womack, G. K.: Ann. N. Y. Acad. Sci. 76:863, 1958. 3. Bunn, P. A., Baltch, A., and Krajnyak, O.: Ibid. 76:109, 1958. 4. Council on Drugs, J.A.M.A. 172:699, 1960

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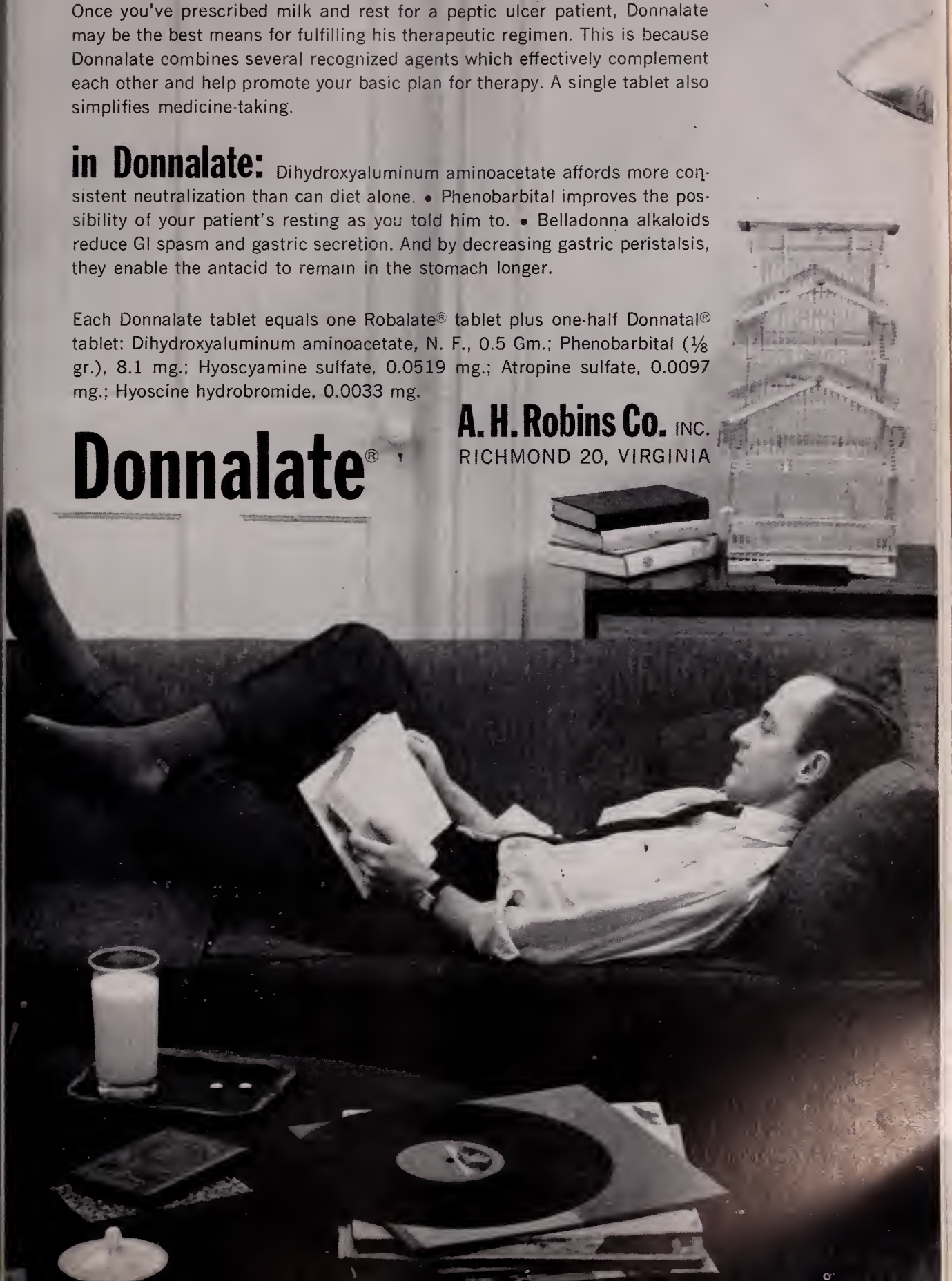
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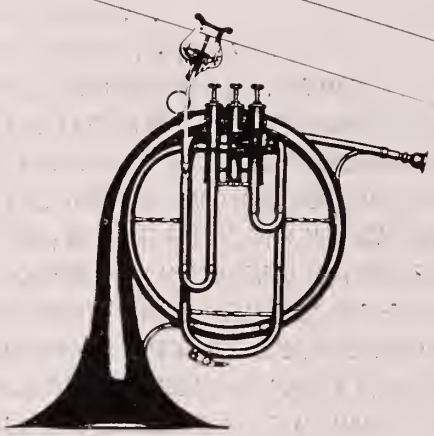
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Rautensin bibliography: 1. WRIGHT, W. T., JR.; POKORY, C., AND FOSTER, T.: AM. PRACT. & DIGEST. TREAT. 7:1992, 1956. 2. SUCKLE, E.: GERIATRICS 11:509, 1956. 3. FINCH, W. J.: J. OKLAHOMA M.A. 50:259, 1957. 4. TERMAN, L. A.: ILLINDIS M. J. 3:67, 1957. 5. GIFFORD, R. W.: J. ARKANSAS M. SOC. 55:31, 1958. 6. FORD, R. V., AND MOYER, J. H.: POSTGRAD. MED. 23:41, 1958.

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Preferably, antibiotic therapy should be based on pretreatment culture of the offending pathogen, but in bacterial pneumonia the problem may well be too pressing to permit the required delay of 24 to 48 hours. A differential diagnosis among bacterial pneumonias, based on such clinical grounds as speed of onset, sepsis and pain may guide the choice of antibiotic for initiation of therapy.

Should clinical judgment dictate that antibiotic therapy be started immediately, at the same time a sputum sample or a subglottic swab can be sent to the laboratory for culture and sensitivity studies. If the response to the first antimicrobial agent proves unsatisfactory, a reasonable basis for changing therapy will then be at hand.

Choosing the Antibiotic

Since therapy must be started at once for bacterial pneumonia, it is advisable to choose a broad-spectrum antibiotic that quickly produces high levels of active agent (e.g., tetracycline phosphate complex, TETREX). Such an antibiotic probably has the best chance of controlling the pathogen, whether it be gram-negative or gram-positive. And if the laboratory report shows that the invading organism is much less sensitive to tetracycline than to other agents, the patient can then be changed to an appropriate antibiotic. If the difference in sensitivity is slight, then the possibility of side effects, sensitization, and toxicity should be evaluated before changing therapy to another antibiotic.

The greatest number of bacterial pneumonias are caused by pneumococci, which respond very well to penicillin, tetracycline, and chloramphenicol. Also, these antibiotics are usually effective against the other gram-positive coccal pneumonias. But penicillin is ineffective against the viral pneumonias and the gram-negative *Hemophilus influenzae* and *Klebsiella pneumoniae*. Although *K. pneumoniae* causes only about 1 to 2 per cent of pneumonia cases on the average,[1] these are apt to be acute and fulminating (Friedländer's pneumonia), with a high mortality rate if not effectively treated. Since pneumococcal pneumonia may be difficult to distinguish clinically from Friedländer's, except by gram-stained sputum smear, it may be wiser to start treatment with an agent also effective against Klebsiella.

Penicillin, however, in addition to having a limited spectrum, also causes many minor and some serious sensitivity reactions. In a recent survey[2] it was found that penicillin produced severe skin reaction. But most important was the observation that anaphylactic shock, with a fatality rate of about 9 per cent, was the most frequent serious reaction. Such severe reactions are almost always associated with parenteral administration.

Tetracycline is also clinically effective in primary atypical pneumonia.[3]

The tetracyclines (e.g., TETREX) have the advantage of a broad range of antimicrobial activity and low toxicity. And in addition, the physician does not have to trouble himself or his patients with repeated blood studies when he prescribes TETREX. Minor reactions such as gastric upsets or mild skin rashes occur occasionally. The most serious side effects are staphylococcal and monilial overgrowth, but these are rare and can be adequately controlled.

No one would deny that appropriate antibiotic therapy has greatly reduced morbidity and saved many lives of patients with bacterial pneumonia. Nevertheless, general supportive measures in the care of patients remain important even today. Especially in the desperately ill patient, antibiotics are not considered as substitutes for the individual evaluation, clinical observation and judgment of the physician.

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Streptococcus; Staphylococcus; Pneumococcus; Gonococcus; Meningococcus; *C. diphtheriae; B. anthracis; E. coli;* Proteus; *A. aerogenes; Ps. aeruginosa; K. pneumoniae;* Shigella; Brucella; *P. tularensis; H. influenzae; T. pallidum;* Rickettsiae; Viruses of psittacosis and ornithosis, lymphogranuloma inguinale, primary atypical pneumonia; *E. histolytica; D. granulomatosis.*

a Some strains are not susceptible.

b Table adapted from Goodman, L. S., and Gilman, A.: The Pharmaceutical Basis of Therapeutics. 2nd edition, New York, The Macmillan Co., 1956, pp. 1322-1323.

References: 1. Wood, W. E., Jr.: In: A Textbook of Medicine. Edited by Cecil, R. L., and Loeb, R. F., 9th edition, Philadelphia, W. B. Saunders Co., 1955, p. 145. 2. Weleb, H.; Lewis, C. H.; Weinstein, H. I., and Boeckman, B. B.: Severe reactions to antibiotics. A nationwide survey. Antibiotic Med. & Clin. Ther. 4:800 (Dec.) 1957. 3. Keefer, C. S.: The choice of an anti-infective agent. In: Drugs of Choice, 1958-1959. Edited by Walter Modell, St. Louis, The C. V. Mosby Co., 1958, p. 135.

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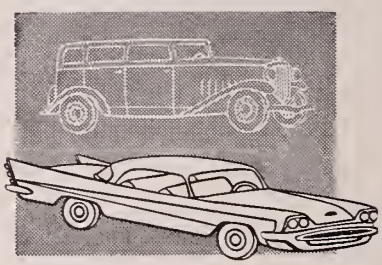


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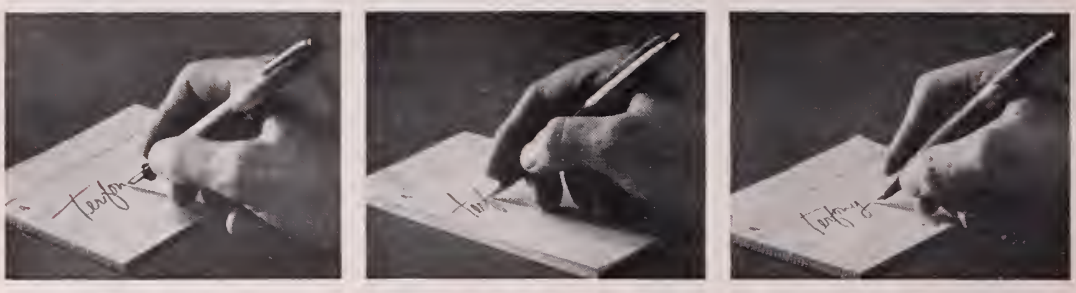
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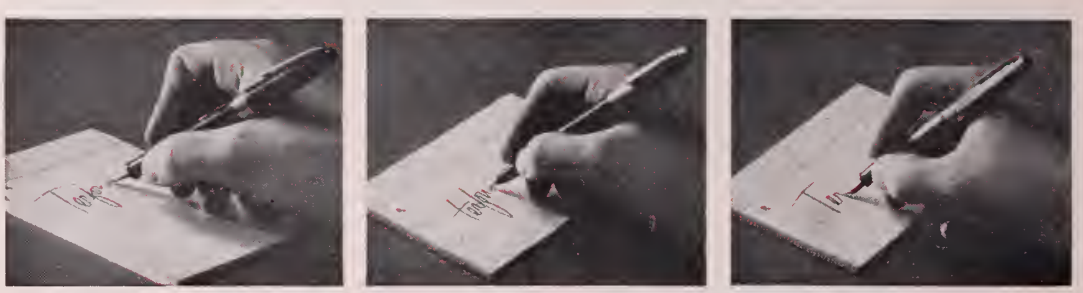
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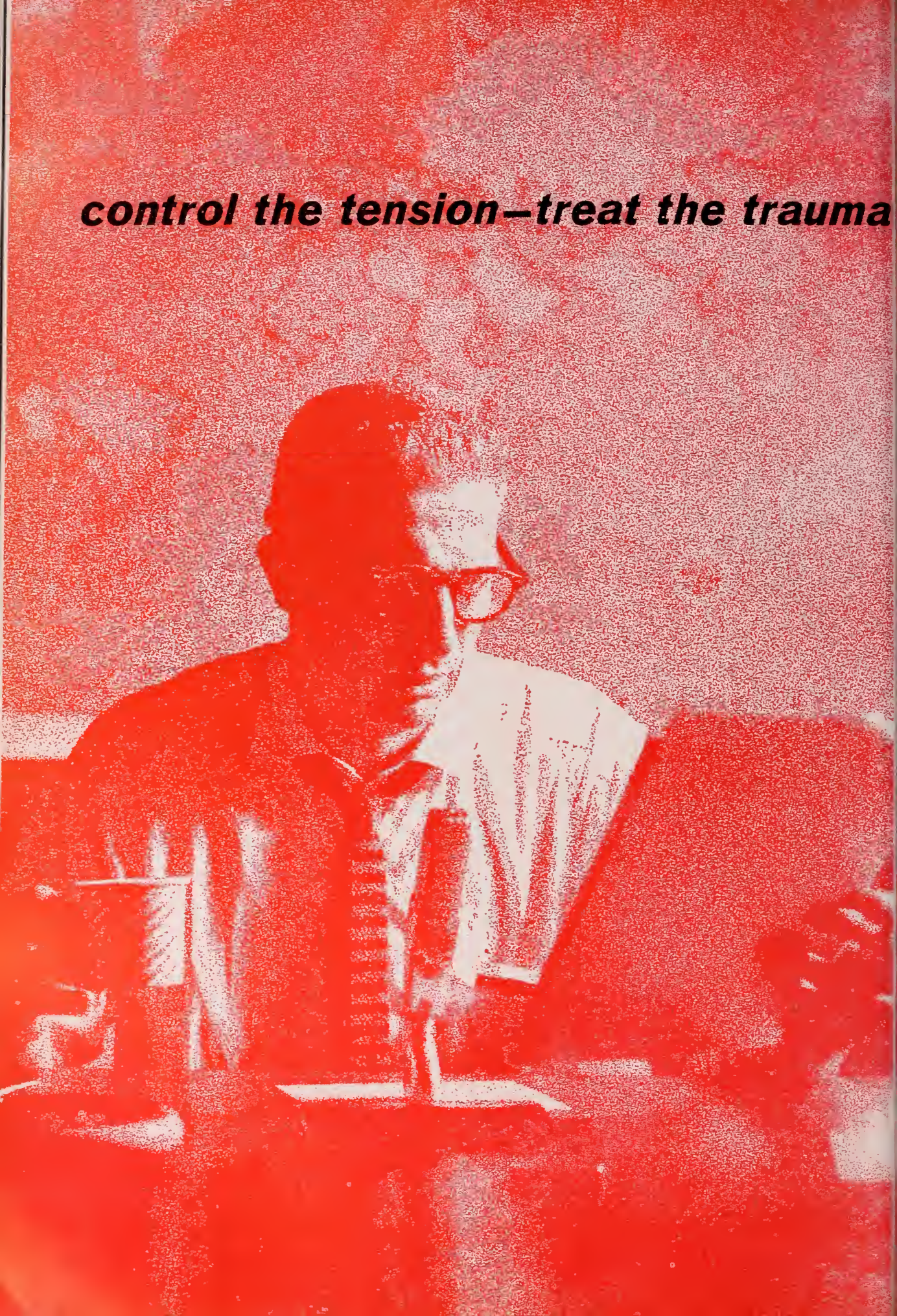
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Friedman, A. P., and Merritt, H. H.: J.A.M.A. *163*:1111 (Mar. 30) 1957.

Fiorinal – available as tablets and *capsules* (new form). *Each contains:* Sandoptal (Allylbarbituric Acid N.F. X) 50 mg. (¾ gr.), caffeine 40 mg. (⅔ gr.), acetylsalicylic acid 200 mg. (3 gr.), acetophenetidin 130 mg. (2 gr.).
Dosage: 1 or 2 every 4 hours, according to need, up to 6 per day.

SANDOZ

allergic nose?



Dimetane® (PARABROMDYLAMINE MALEATE) Works!

with side effects as few as placebo

—New England J. Med. *261*:478, 1959 (Schiller, I. W. and Lowell, F. C.)

Dimetane works with an effectiveness of 91% in respiratory allergies —NEW YORK J. MED. *59*:3060, 1959 (Fuchs, A. M. and Maurer, M. L.). In allergic and pruritic dermatoses the effectiveness rate of Dimetane is 94.6% —ANTIBIOTIC MED. & CLIN. THERAPY *6*:275, 1959 (Lubowe, I. I.). The A. M. A. Council on Drugs characterizes Dimetane as demonstrating "...a high order of antihistaminic effectiveness and a low incidence of side effects." —J.A.M.A. *170*:194, 1959.

for your next allergic patient ℞ DIMETANE Extentabs® (12 mg.), Tablets (4 mg.), Elixir (2 mg./5 cc.), new DIMETANE-TEN Injectable (10 mg./cc.) or new DIMETANE-100 Injectable (100 mg./cc.).

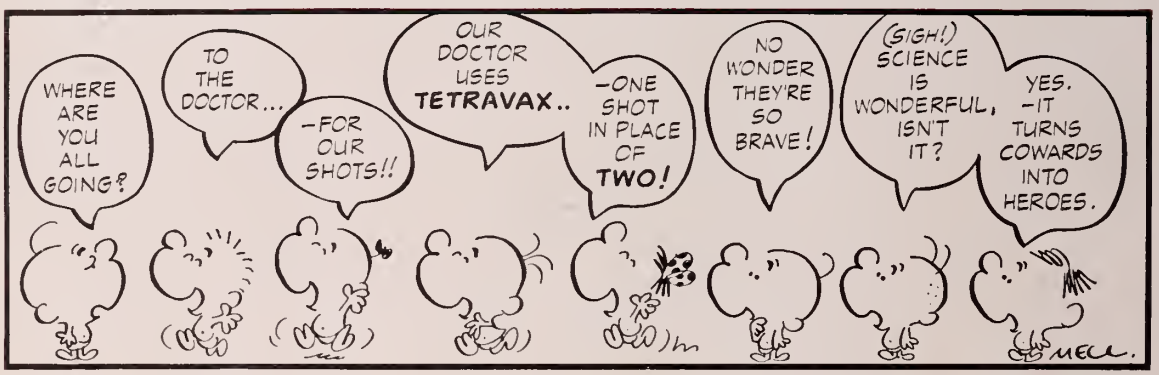
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DIPHTHERIA AND TETANUS TOXOIDS WITH PERTUSSIS AND POLIOMYELITIS VACCINES

now you can immunize against more *diseases...with* fewer *injections*

Dose: 1 cc.
Supplied: 9 cc. vials in *clear plastic* cartons. Package circular and material in vial can be examined without damaging carton. Expiration date is on vial for checking even if carton is discarded.



For additional information, write Professional Services, Merck Sharp & Dohme, West Point, Pa.

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A.P.C. WITH DEMEROL® (30 mg.)

1 or 2 tablets three or four times daily.

Considerably more effective than A.P.C. with codeine.

NARCOTIC BLANK REQUIRED.

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'ACTIFED'®

Decongestant / Antihistamine

THE POTENTIATED DECONGESTANT



provides symptomatic relief of nasal congestion and rhinorrhea of allergic or infectious origin

Many patients whose symptoms are inadequately controlled by decongestants or antihistamines alone respond promptly and favorably to 'ACTIFED'.

'ACTIFED' contains:	in each Tablet	in each tsp. Syrup
'Actidil'® brand Triprolidine Hydrochloride	2.5 mg.	1.25 mg.
'Sudafed'® brand Pseudoephedrine Hydrochloride	60 mg.	30 mg.

safe and effective for patients of all ages suffering from respiratory tract congestion

DOSAGE

	TABLETS	SYRUP (5 cc. tsp.)	
Adults and older children	1	2	three times daily
Children 4 months to 6 years of age	½	1	
Infants through 3 months	–	½	

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Q. *When you want to reduce serum cholesterol and maintain it at a low level, is medication m realistic than dietary modifications?*

A. *Maintenance of lowered cholesterol concentration in the blood is a life-long problem. It is usually preferable, therefore, to try to obtain the desired results through simple dietary modification. This spares the patient added expense and permits him meals he will relish.*

The modification is based on a diet to maintain optimum weight plus a judicious substitution of the poly-unsaturated oils for the saturated fats. One very simple part of the change is to cook the selected foods with poly-unsaturated Wesson. In the prescribed diet, this switch in type of fat will help to lower blood serum cholesterol and help maintain it at low levels. The use of Wesson permits a diet planned around many favorite and popular foods. Thus the patient finds it a pleasant, easy matter to adhere to the prescribed course.



Where a vegetable (salad) oil is medically recommended for a cholesterol depressant regimen, Wesson is unsurpassed by any readily available brand.

Uniformity you can depend on. Wesson has a poly-unsaturated content better than 50% . Only the lightest cottonseed oils of highest iodine number are selected for Wesson. No significant variations are permitted in the 22 exacting specifications required before bottling.

Wesson satisfies the most exacting appetites. To effective, a diet must be *eaten* by the patient. majority of housewives prefer Wesson particularl the criteria of odor, flavor (blandness) and lightne color. (Substantiated by sales leadership for 59 y and reconfirmed by recent tests against the leading brand with brand identification removed, an a national probability sample.)

Chicken, grilled with homemade Wesson barbecue sauce, is low in saturated fat—and delicious eating. It gives longer lasting satisfaction.



FREE *Wesson recipes, available in quantity for your patients, show how to prepare meats, seafoods, vegetables, salads and desserts with poly-unsaturated ble oil. Request quantity needed from he Wesson People, Dept. N., 210 Baronne St., New Orleans 12, La.*

Wesson's Important Constituents

Wesson is 100% cottonseed oil . . . winterized and of selected quality

Linoleic acid glycerides (poly-unsaturated)	50-55%
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Total unsaturated	70-75%
Palmitic, stearic and myristic glycerides (saturated)	25-30%
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Total tocopherols	0.09-0.12%

Never hydrogenated—completely salt free

Each pint of Wesson contains 437-524 Int. Units of Vitamin E



in one preparation

the answer to your
three most important
requirements in
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For a dependable and effective means of treating non-specific leukorrhea

For adjunctive therapy in Trichomonas Vaginalis vaginitis and other specific infections

For personal cleanliness and the prevention of irritation and inflammation

TRICHOTINE is the first major douche to contain sodium lauryl sulfate, a detergent of the highest order of efficiency. TRICHOTINE penetrates and dissolves the viscid film covering the vaginal mucosa; gets down in the rugal folds, carrying medication directly to the mucosa and the invading organisms.

TRICHOTINE is a potent bactericide and fungicide, penetrating the walls

TRICHOTINE®





of many micro-organisms. "The douche solution is an effective agent against Trichomonas Vaginalis, Monilia Albicans, anaerobic organisms including a potent strain of streptococci that sometimes cause severe infections, and other non-specific vaginal micro-organisms."[1]

TRICHOTINE actually favors epithelial growth and healing, and the relief it affords from pruritis is quite striking.

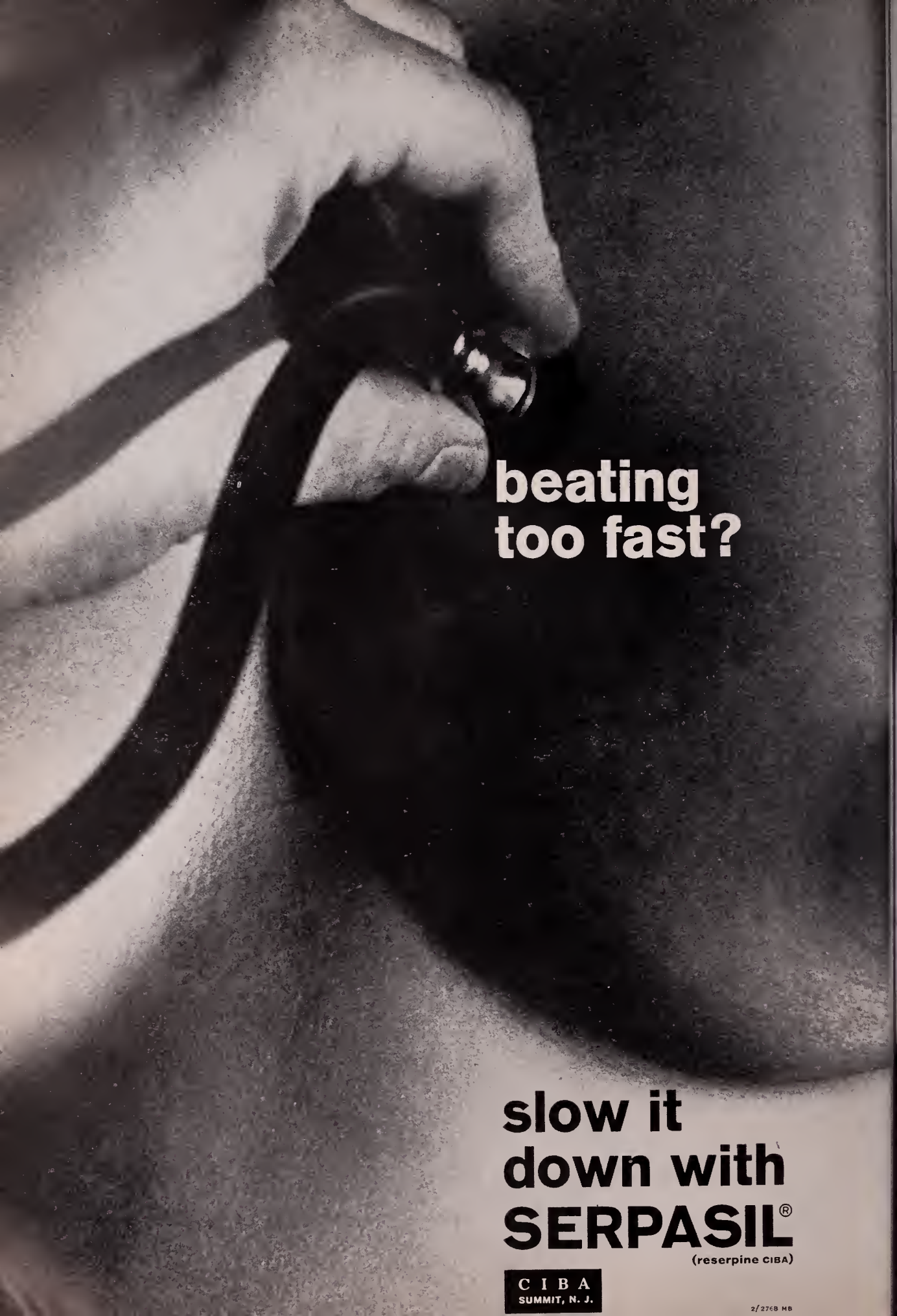
For personal cleanliness, especially as a post-coital and post-menstrual douche, TRICHOTINE is designed to meet all the requirements of feminine hygiene. As an effective cleanser for office use, or for treatment, or for routine home douching, TRICHOTINE will prove satisfactory to you and its soothing, refreshing action will be reassuring to your patients.

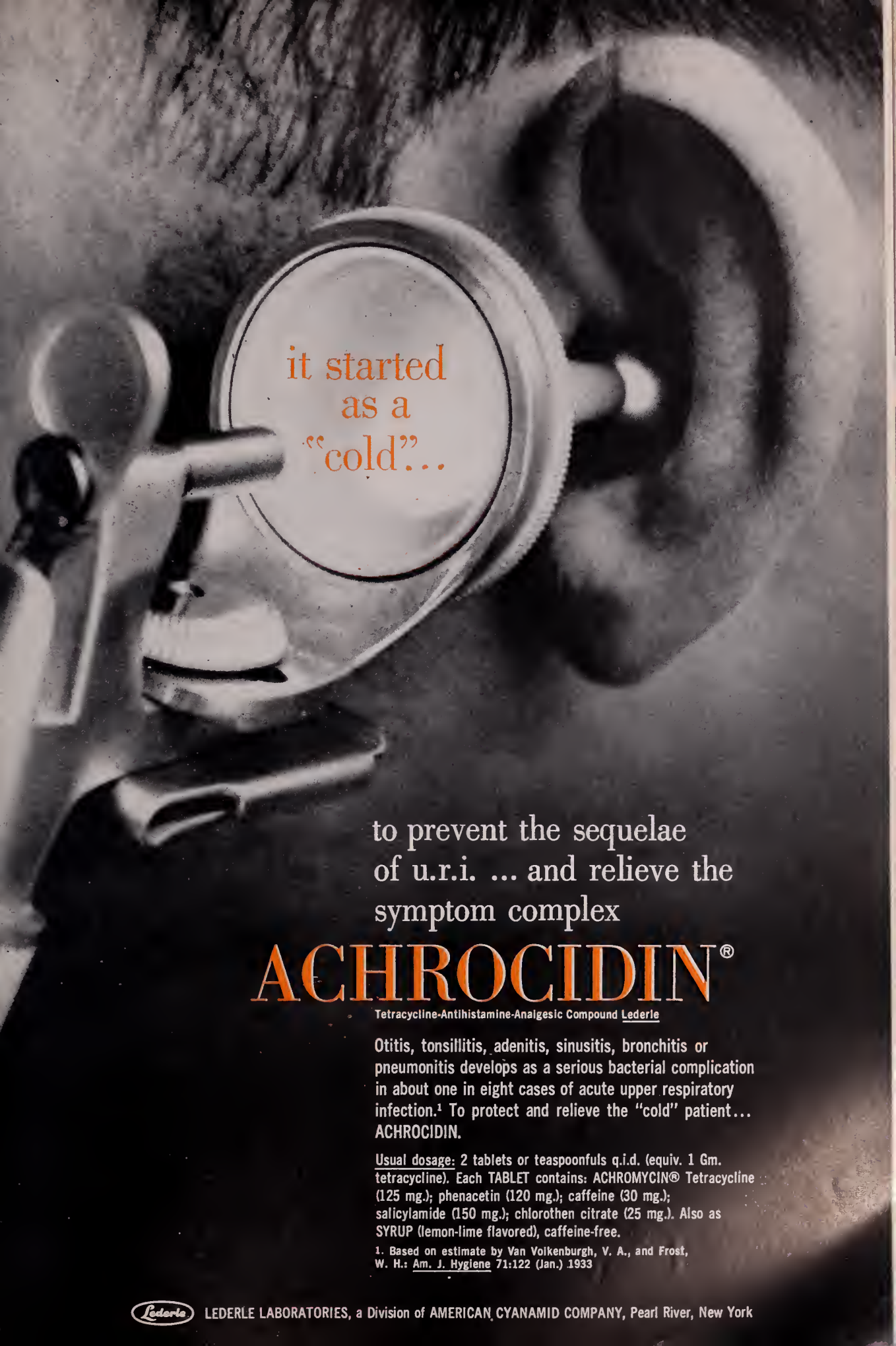
1. Karnaky, K.J.: Med. Record and Annals, Houston 46:296 (Nov. 1952).

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slow it
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to prevent the sequelae of u.r.i. ... and relieve the symptom complex

ACHROCIDIN®

Tetracycline-Antihistamine-Analgesic Compound Lederle

Otitis, tonsillitis, adenitis, sinusitis, bronchitis or pneumonitis develops as a serious bacterial complication in about one in eight cases of acute upper respiratory infection.[1] To protect and relieve the "cold" patient... ACHROCIDIN.

Usual dosage: 2 tablets or teaspoonfuls q.i.d. (equiv. 1 Gm. tetracycline). Each TABLET contains: ACHROMYCIN® Tetracycline (125 mg.); phenacetin (120 mg.); caffeine (30 mg.); salicylamide (150 mg.); chlorothen citrate (25 mg.). Also as SYRUP (lemon-lime flavored), caffeine-free.

1. Based on estimate by Van Volkenburgh, V. A., and Frost, W. H.: Am. J. Hygiene 71:122 (Jan.) 1933

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no irritating crystals[1] · uniform concentration in each drop[2]

STERILE OPHTHALMIC SOLUTION

NEO-HYDELTRASOL®

PREDNISOLONE 21-PHOSPHATE-NEOMYCIN SULFATE

2,000 TIMES MORE SOLUBLE THAN PREDNISOLONE OR HYDROCORTISONE

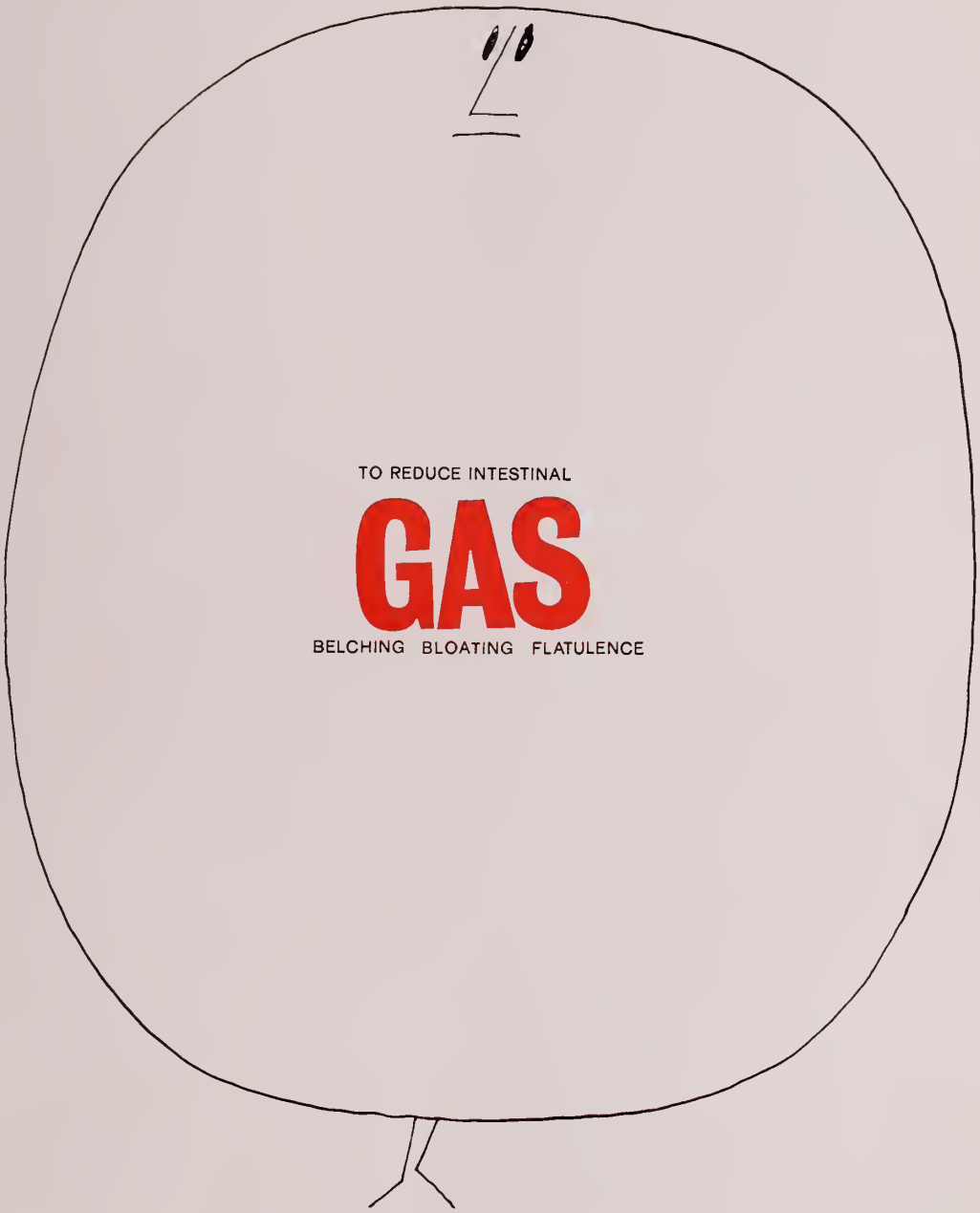
"The solution of prednisolone has the advantage over the suspension in that no crystalline residue is left in the patient's cul-de-sac or in his lashes . . . . The other advantage is that the patient does not have to shake the drops and is therefore sure of receiving a consistent dosage in each drop."[2]

1. Lippmann, O.: Arch. Ophth. **57**:339, March 1957
2. Gordon, D.M.. Am. J. Ophth. **46**:740, November 1958.

supplied: 0.5% Sterile Ophthalmic Solution NEO-HYDELTRASOL (with neomycin sulfate) and 0.5% Sterile Ophthalmic Solution HYDELTRASOL*. In 5 cc. and 2.5 cc dropper vials. Also available as 0.25% Ophthalmic Ointment NEO-HYDELTRASOL (with neomycin sulfate) and 0.25% Ophthalmic Ointment HYDELTRASOL. In 3.5 Gm. tubes.

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A biochemical compound used to diminish intestinal gas in healthy persons and those patients having digestive disorders ■ **KANULASE**

Each Kanulase tablet contains Dorase,* 320 units, combined with pepsin, N.F., 150 mg.; glutamic acid HCl, 200 mg.; pancreatin, N.F., 500 mg.; oxbile extract, 100 mg. Dosage: 1 or 2 tablets at mealtime. Supplied: Bottles of 50 tablets.

*CONC'D BRAND OF CELLULASE, EXPRESSED AS DIGESTIVE ACTIVITY UNITS

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The first synthetic penicillin available for general clinical use

FOR YOUR NEXT PATIENT WHERE PENICILLIN IS INDICATED

SYNCILLIN

PEAK BLOOD LEVELS HIGHER THAN POTASSIUM PENICILLIN V

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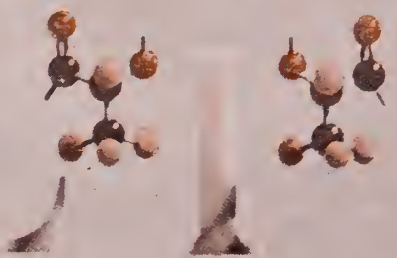
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ANTIBIOTIC ACTIVITY DIRECTLY PROPORTIONAL TO ORAL DOSE

REDUCED RATE OF INACTIVATION BY STAPH PENICILLINASE

SOME STAPH STRAINS MORE SENSITIVE TO SYNCILLIN IN VITRO

BRISTOL





FOR HIGHLY EFFECTIVE THERAPY OF THE LARGE VARIETY OF INFECTIONS CAUSED BY SUSCEPTIBLE PATHOGENS...NEW

SYNC

Significance of complementary action of isomers in SYNCILLIN

The antibiotic effect of the clinically available mixture, SYNCILLIN, is greater than that of either of its two component isomers alone against many important pathogens, including some penicillin-resistant staphylococci. This phenomenon has been described as *Isomeric Complementarity*.

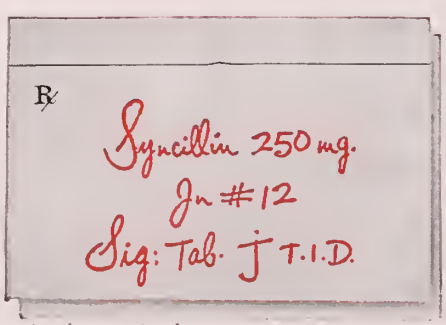
Significance of higher blood levels with SYNCILLIN

Higher blood levels may be of value with organisms of only moderate penicillin sensitivity where doubling the blood concentration may be essential for effective bactericidal action. In addition, these higher levels may be necessary where there is infection in areas with a poor blood supply. Under these circumstances a higher blood concentration may provide the increased diffusion pressure required to deliver adequate amounts to the tissue. Also, antibiotic activity of SYNCILLIN is directly proportional to oral dosage. Increasing the dosage may, therefore, enhance the drug's effectiveness in certain cases.

Efficacy of SYNCILLIN against staphylococci and other resistant organisms

Studies have shown that SYNCILLIN is effective *in vitro* against a higher percentage of hospital "staph" strains, than penicillin G and penicillin V.[1,2] Therefore, if clinical judgment indicates the use of penicillin, SYNCILLIN might be expected to be somewhat more effective. However, since some strains are still resistant to SYNCILLIN as well as to the other penicillins, cultures and sensitivity tests should be performed where indicated by clinical judgment.

There have recently been reports of decreased efficacy of penicillin in streptococcal[3] and gonococcal[4,5] infections. The emergence of penicillin-resistant gonococci appears to be associated with an increase in the incidence of gonorrhea all over the world. When a less sensitive strain is encountered the higher blood levels produced by SYNCILLIN may be most helpful.



major therapeutic advantages accompany molecular asymmetry

LLIN™

potassium phenethicillin (POTASSIUM PENICILLIN-152)

Relation of intermittent high blood levels of SYNCILLIN to antibacterial efficacy

SYNCILLIN, like all clinically available penicillins, is bactericidal. Periodic high blood concentrations may be sufficient to permit complete eradication of sensitive pathogens. According to Eagle,[6] "Soon after penicillin attains effective concentrations, the bacteria cease multiplying; and the bacteriostatic effect persists for a number of hours after penicillin has fallen to concentrations that are wholly ineffective. . . . The therapeutic significance of this postpenicillin recovery period is enhanced by the fact that the recovering bacteria, damaged but not killed by the previous exposure to penicillin, are abnormally susceptible to the host defenses. In consequence, the bactericidal process *in vivo* continues for many hours after the drug itself has fallen to ineffective concentrations."

Reduced rate of inactivation of SYNCILLIN by staph penicillinase

Bacterial resistance to penicillin has been attributed to the action of penicillin-inactivating enzymes produced by the invading organisms. SYNCILLIN is less affected by staphylococcal penicillinase than either of its component isomers. Further, SYNCILLIN is shown to be more slowly inactivated by this enzyme than penicillin V or penicillin G. Penicillinase from *B. cereus* likewise inactivates SYNCILLIN less rapidly than penicillin V or G.

Indications: SYNCILLIN is recommended in the treatment of infections caused by pneumococci, streptococci, gonococci, corynebacteria, and penicillin-sensitive staphylococci. In addition, SYNCILLIN is effective *in vitro* against certain strains of staphylococci resistant to other penicillins. SYNCILLIN, like other oral penicillins, is not recommended at the present time in deep-seated or chronic infections, subacute bacterial endocarditis, meningitis, or syphilis.

Dosage: 125 mg. or 250 mg. three times daily, depending on the severity of infection. Larger doses (e.g., 500 mg. t.i.d.) may be used for more severe infections. SYNCILLIN may be administered without regard to meals. Beta hemolytic streptococcal infections should be treated with SYNCILLIN for at least ten days.

Precautions: At the present time it is not possible to draw definite conclusions regarding the incidence of allergenicity to SYNCILLIN or its cross-allergenicity with natural penicillins. *Therefore, the usual precautions for oral penicillin therapy should always be observed.* Patients with histories of asthma, hay fever, urticaria, or previous reactions to penicillin should be watched with special care. Administration of oral penicillin, in rare instances, may provoke acute anaphylaxis, particularly in penicillin-sensitive individuals.

Diarrhea has been reported occasionally following heavy dosage. If this occurs, lengthen the interval between dosages.

If superinfection occurs during therapy, appropriate measures should be taken. Since some strains of staphylococci are resistant to SYNCILLIN as well as to other penicillins, cultures and sensitivity tests should be performed where indicated by clinical judgment. As is true with all antibiotics, clinical response does not always correlate with laboratory bacterial sensitivity reports.

Supply: 125 and 250 mg. tablets, bottles of 25 and 100. 125 mg. powder for oral solution, 60 ml. vials.

References: 1. Wright, W. W.: Microbiology Report to Bristol Laboratories Inc. 2. Morigi, E. M. E.; Wheatley, W. B., and Albright, H.: Paper presented at the Seventh Antibiotic Symposium, November 4-6, 1959, Washington, D.C. 3. Editorial: New England J. Med. **261**:305 (Aug. 6) 1959. 4. King, A.: Lancet **1**:651 (March 29) 1958. 5. Epstein, E.: J.A.M.A. **169**:1055 (March 7) 1959. 6. Eagle, H. and Musselman, A. D.: J. Bact. **58**:475, 1949.

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in infectious disease[17,22,30,]
in arthritis[18,19,20,]
in hepatic disease[2,3,4,5,]
in malabsorption syndrome[1,2,6,]
in degenerative disease[6,7,19,20,]
in cardiac disease[23,28,29,38,]
in dermatitis[24,3]
in peptic ulcer[8,21,3]
in neuroses & psychiatric disorders[25,2]
in diabetes mellitus[31,32,33,3]
in alcoholism[9,11,35,37,3]
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in osteoporosis[13,19,2]
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in female climacteric[12,34]

Patients with chronic disease deserve the nutritional support provided by

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11 vitamins, 8 minerals
clinically-formulated and potency
protected to provide
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1-41 a list of the above references will be supplied on request.

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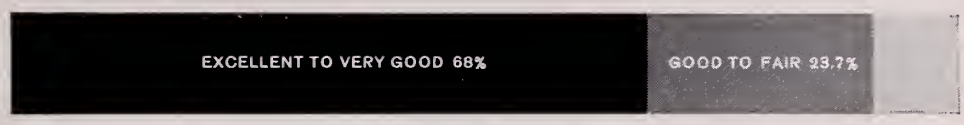
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Squibb Quality—the Priceless Ingredient

relieves both stiffness and pain with safety... sustained effect

In 100 consecutive patients with the low back syndrome, Kestler[1] reported that particularly gratifying was the ability of SOMA "to relax muscular spasm, relieve pain, and restore normal movement, thus speeding recovery in a large majority of the patients."

RESULTS WITH SOMA IN THE LOW BACK SYNDROME*



*Investigators' reports to the Medical Department, Wallace Laboratories. (Total of 278 cases)

NOTABLE SAFETY—extremely low toxicity; no known contraindications; side effects are rare; drowsiness may occur, usually at higher dosage

RAPID ACTION—starts to act quickly **SUSTAINED EFFECT**—relief lasts up to 6 hours

EASY TO USE—usual adult dosage is one 350 mg. tablet 3 times daily and at bedtime

SUPPLIED—as white, coated, 350 mg. tablets, bottles of 50; also available for pediatric use: 250 mg., orange capsules, bottles of 50

1. Kestler, O.: In The Pharmacology and Clinical Usefulness of Carisoprodol, Wayne State University Press, Detroit, 1959. 2. Berger, F. M.; Kletzkin, M.; Ludwig, B. J.; Margolin, S., and Powell, L. S.: J. Pharm. Exp. Ther. *127*:66 (Sept.) 1959. 3. Spears, C. E. and Phelps, W. M.: Arch. Pediat. *76*:287 (July) 1959. 4. Phelps, W. M.: Arch. Pediat. *76*:243 (June) 1959. 5. Friedman, A. P.; Frankel, K., and Fransway, R. L.: Papers presented at Scientific Meeting, New York State Society of Industrial Medicine, Inc., New York, Sept. 30, 1959. 6. Kuge, T.: Unpublished reports. 7. Ostrowski, J. P.: Orthopedics 2:7 (Jan.) 1960.

Literature and samples on request

Also available on request: The Pharmacology and Clinical Usefulness of Carisoprodol, Wayne State University Press, Detroit, 1959. (185 pages)

SOMA™

(carisoprodol Wallace)

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DOES YOUR PRESENT ANTICHOLINERGIC REALLY



The test—you might say the acid test—of an anticholinergic is simple: will it protect your patient from hyperacidity around the clock, **even while he sleeps.** The weakness of t.i.d. or q.i.d. preparations is well recognized; but even some "b.i.d." encapsulations may be unreliable. McHardy, for instance, found a "widely variable duration of action, definitely less than that anticipated" in the "sustained," "delayed," and "gradual release" anticholinergics he studied.[1]

COMPARE THE DATA ON ENARAX . . . the new combination of an **inherently** long-acting anticholinergic (oxyphencyclimine) and Atarax, the non-secretory tranquilizer. Note the effectiveness of oxyphencyclimine:

OBSERVE THE OXYPHENCYCLIMINE REPORTS . . .

McHardy: "[Oxyphencyclimine] has proved to be an excellent sustained-action anticholinergic in our study of this agent over a period of eighteen months."[1]

Kemp: ". . . for the majority of patients, one tablet every 12 hours provided adequate control. This characteristic long action . . . may constitute an advantage of this drug as compared to coated 'long-acting' preparations of other compounds."[3]

Add Atarax to this 12-hour anticholinergic. The resulting combination—ENARAX—now gives relief from emotional stress, in addition to a reduction of spasm and acid. Atarax does not stimulate gastric secretion. No serious adverse clinical reaction has ever been documented with Atarax.

LOOK AT THE RESULTS WITH ENARAX[4,5]:

Does the medication you now prescribe assure you of all these benefits? If not, why not put your next patient with peptic ulcer or G.I. dysfunction on therapy that **does.**

ENARAX®

(oxyphencyclimine plus ATARAX®)

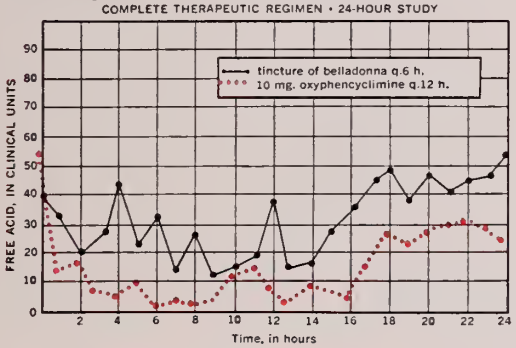
A SENTRY FOR THE G.I. TRACT

PROVIDE CONTINUOUS CONTROL OF ACID SECRETION?



"Prolonged periods of achlorhydria" after 10 mg. oxyphencyclimine q. 12 h.[2]

MEAN GRAPH OF GASTRIC ACIDITY IN 4 PATIENTS RECEIVING COMPLETE THERAPEUTIC REGIMEN • 24-HOUR STUDY



Clinical Diagnosis: Peptic Ulcer—Gastritis—Gastroenteritis—Colitis—Functional Bowel Syndrome—Duodenitis—Hiatus Hernia (symptomatic)—Irritable Bowel Syndrome—Pylorospasm—Cardiospasm—Biliary Tract Dysfunctions—and Dysmenorrhea.

Clinical Results: Effective in over 92% of cases.

As for Safety: "Side reactions were uncommon, usually no more than dryness of the mouth...."[4]

Each ENARAX tablet contains:

Oxyphencyclimine HCl ..... 10 mg.
Hydroxyzine (ATARAX®) ..... 25 mg.

Dosage: One-half to one tablet twice daily—preferably in the morning and before retiring. The maintenance dose should be adjusted according to therapeutic response. Use with caution in patients with prostatic hypertrophy and with ophthalmological supervision only in glaucoma.

Supplied: In bottles of 60 black-and-white scored tablets.

References: 1. McHardy, G., et al.: J. Louisiana M. Soc. 111:290 (Aug.) 1959. 2. Steigmann, F.: Study conducted at Cook County Hospital, Chicago, Illinois, in press. 3. Kemp, J. A.: Antibiotic Med. & Clin. Therapy 6:534 (Sept.) 1959. 4. Leming, B. H., Jr.: Clin. Med. 6:423 (Mar.) 1959. 5. Data in Roerig Medical Department files.

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make them measure up



Incremin® with iron Syrup

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...elp restore the normal blood picture—iron as ferric pyrophosphate to restore or maintain normal hemoglobin.

boost appetite and energy—vitamins . . . B_1, B_6 and B_{12}.

...pgrade low-grade protein—cereals and other low ...rotein favorites of children, upgraded by l-Lysine, work with meat and other top protein to build stronger bodies.

tastes good! Each daily cherry-flavored teaspoonful dose (5 cc.) contains:

l-Lysine HCl	300 mg.
Vitamin B_{12} Crystalline....	25 mcgm.
Thiamine HCl (B_1)	10 mg.
Pyridoxine HCl (B_6)	5 mg.
Ferric Pyrophosphate (Soluble)	250 mg.
Iron (as Ferric Pyrophosphate)	30 mg.
Sorbitol	3.5 Gm.
Alcohol	0.75%

Bottles of 4 and 16 fl. oz.

Lederle LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

reaches all nasal and paranasal membranes *systemically*[1]



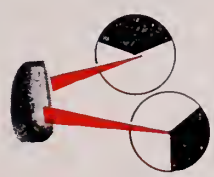
Pharmacologically balanced formula for prompt symptomatic relief

- in nasal and paranasal congestion
- in sinusitis and postnasal drip
- in allergic reactions of the upper respiratory tract

Triaminic*[2,3] *is safer and more effective than topical medication

- transported systemically to *all* respiratory membranes
- provides longer-lasting relief
- presents no problem of rebound congestion
- avoids "nose drop addiction"

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1. Fabricant, N. D.: E.E.N.T. Monthly *37*:460 (July) 1958.
2. Lhotka, F. M.: Illinois M. J.: *112*:259 (Dec.) 1957.
3. Farmer, D. F.: Clin. Med. *5*:1183 (Sept.) 1958.

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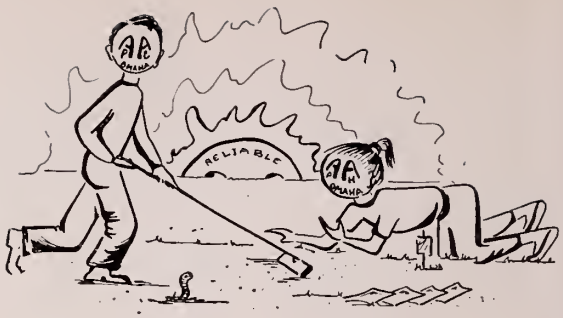
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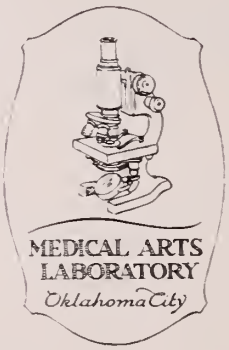
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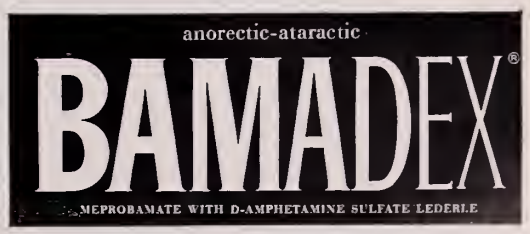
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Index to Advertisers

Ames Company, Inc. ... inside back cover
Arlington Hotel ... lxiii

Beverly Hills Clinic ... lxxiii
Bone and Joint Hospital ... lxvii

George A. Breon & Co. ... xxii
Bristol Laboratories, Inc. ... xx and xxi, xxvii, l, liii
Burroughs-Wellcome & Company, Inc. ... xli

Ciba ... vi, xlvi
Coca Cola Bottlers ... lxiii
Corn Products Refining Co. ... xlii and xliii
Coyne Campbell Sanitarium ... lxvii
Credit Service ... lxiii

The Fesler Company, Inc. ... xliv and xlv

Geigy ... xvi, xxvi
Goldfain Laboratory ... lxviii

J. E. Hanger ... lxi

Drs. Lain, Lamb and Jones ... lxviii
Lederle Laboratories ... viii and ix, 250 and 251, xxxi, xxxiv and xxxv, xlvii, lviii, lxi, lxii, lxiv, lxxiii
Eli Lilly & Company ... xiv

Medical Arts Laboratory ... lxvi
S. E. Massingill Company ... xii
Melton Co., Inc. ... lxiv
Merch Sharp and Dohme, Inc. ... iv, xxxviii, xlviii
McAlester Clinic ... lxix
Midwest Surgical Supply Co., Inc. ... lxxii

Neurological Hospital ... lxxiii
North American Company ... xxx

Oklahoma Allergy Clinic ... lxix
Osler Radioisotope Laboratory ... lxxii

Parke Davis and Company ... inside front and iii
Pfizer Laboratories ... xviii, xxix
Physicians Casualty & Health Assn. ... lx
Physicians Laboratory ... lxxii
Pitman-Moore Company ... xxviii

A. H. Robins Co., Inc. ... xxiii, xxxvii
Rock Haven Hospital ... lxxiii
Roerig ... x, xix, lvi and lvii

St. Paul Companies ... lxi
Sandoz ... xxxvi
Sardeau, Inc. ... xvii
G. D. Searle & Company ... 215
Schering Corporation ... xiii, lx, lxii, lxv
Seven-Up Bottlers ... lxiv
Smith-Dorsey ... xxiv, xxxiii, xlix, lix
Smith, Kline & French Laboratories ... back cover
Springer Clinic ... lxx
E. R. Squibb and Sons ... xxv, xxxii, liv
Stout Clinical Laboratory ... lxx
Sugg Clinic ... lxxi

Terrell's Laboratories ... lxxii
Tulsa Clinic ... lxxi
S. J. Tutag & Co. ... lxv

Wallace Laboratories ... xi, insert, lv
Winthrop Laboratories ... vii, xxxix, insert

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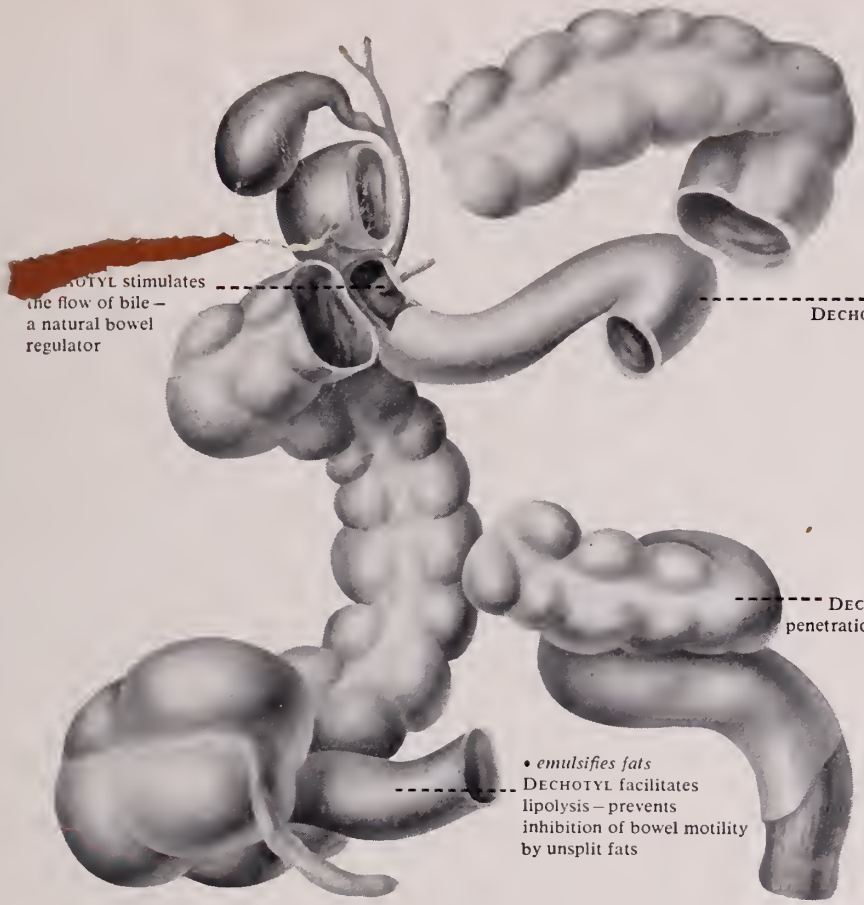
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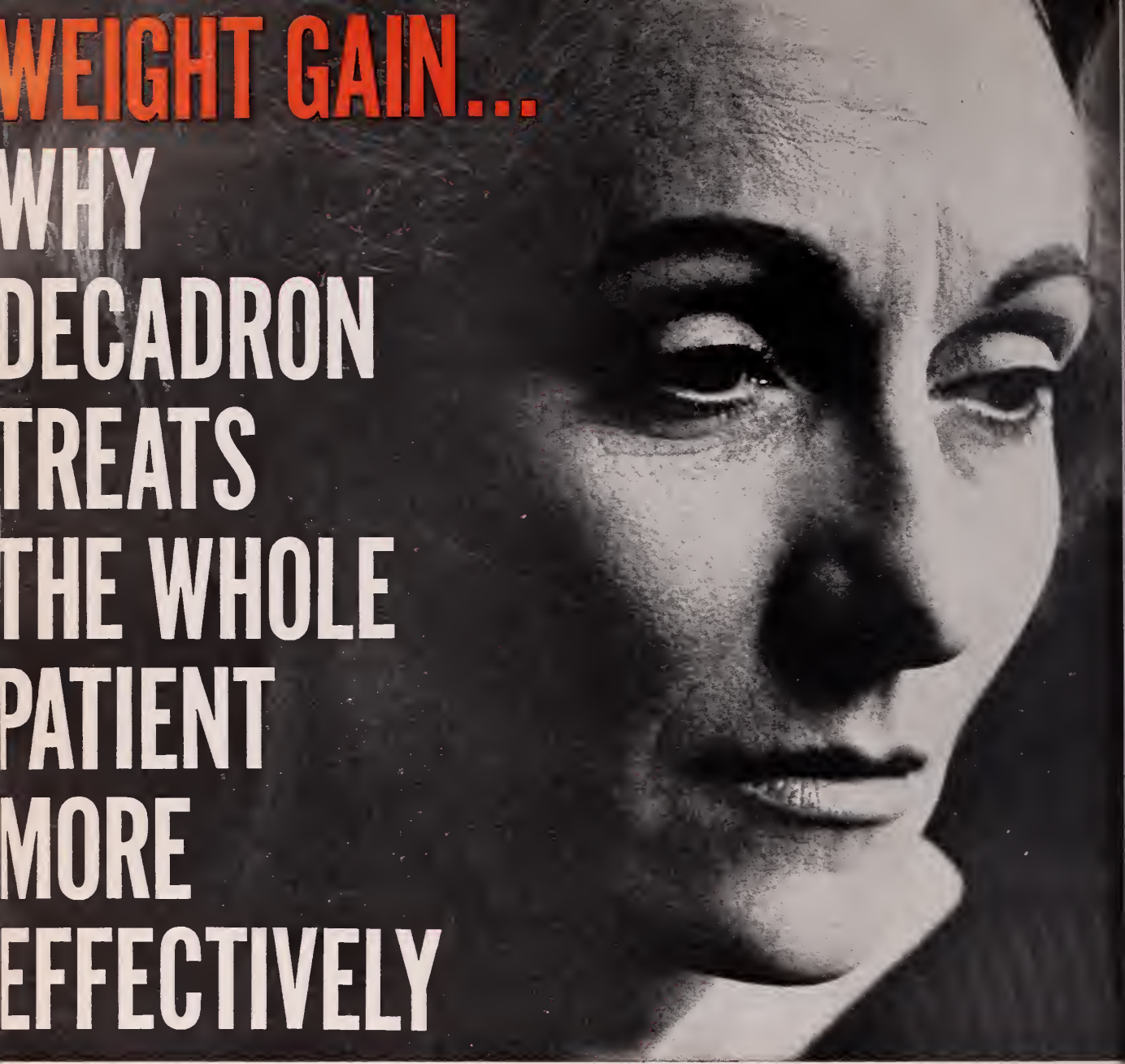
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CONTENTS

EDITORIALS

The Continuous Education of A Physician 259
President's Letter 390

COLLOQUY ON FLUID, ELECTROLYTE AND NUTRITIONAL BALANCE

Introduction 262
Table of Contents 263
Participants 264
The Renal Regulation of Acid-Base Balance: A Summary of Current Views 265
The Relations of Potassium Depletion to Renal Structure and Function 273
Aldosterone and Potassium Metabolism 277
Acute Renal Insufficiency 282
Electrolyte Alterations in Surgical Patients 290
A Practical Guide to Fluid Therapy in Burns 296
The Practice of Fluid Therapy 303
Use of Osmotic Diuretics in Surgery 308
The Problem of Water Intoxication in Pediatrics and Surgery 314
Central Nervous System Factors in Water and Electrolyte Metabolism 319
Stress: Its Effect Upon Fluid and Electrolyte Metabolism 327
Role of the Connective Tissue System in Water and Electrolyte Metabolism 333
The Metabolic Response to Injury 342
Clinical Aspects of Magnesium Metabolism 354
Magnesium and Calcium Interrelationships 359
Clinical Studies in Patients Receiving Long-Term Infusions of a Fat Emulsion 367
Physiological Observations on Fat Transport Utilizing an Intravenous Emulsion 373
The Use of Fat Emulsion, Alcohol and Protein Hydrolysates in Surgical Patients 385

NEWS

Walter E. Brown, M.D., OSMA President 392
Clinton Gallaher, M.D., OSMA President-Elect 392
Seven Hundred Attend 54th Annual Meeting 393
The 109th Annual AMA Meeting: A Forum for 2,000 Top Medical Men . 395
Ringling Student Wins Governor's Essay Contest 397
Carcinoma of the Skin Subject of May Symposium 398
Wallis S. Ivy, M.D., Receives Double Honor 399
AMA, AHA Tighten Up on Foreign Grads 400
"M.D., USA" To Be Seen May 27 400
Armed Forces Will Call Deferred Physicians 401
Nuclear Society Will Meet in Colorado 401
Coming Meetings 402
Auxiliary News 403
Twenty-five Years Ago 404
Miscellaneous Ads xvii, Index to Advertisers lxvi

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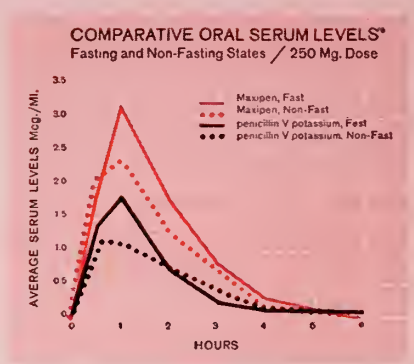
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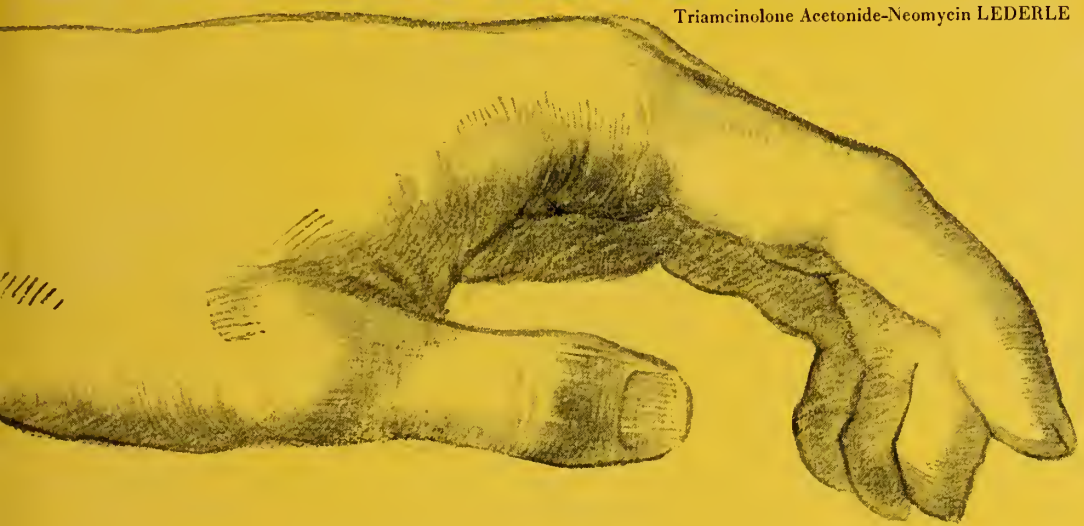
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References: 1. Kanof, N. B., and Blau, S.: *New York J. Med.* 59:2184 (June 1) 1959.
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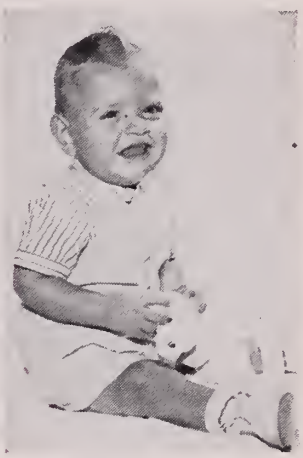
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1. Griffith, R. S.: Comparison of Antibiotic Activity in Sera Following the Administration of Three Different Penicillins, Antibiotic Med. & Clin. Therapy, 7:No. 2 (February), 1960.

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Editorial

The Continuous Education Of A Physician

"The want of energy is one of the main reasons why so few persons continue to improve in later years. They have not the will, and do not know the way. They 'never try an experiment' or look up a point of interest for themselves; they make no sacrifices for the sake of knowledge; their minds, like their bodies, at a certain age become fixed."

Jowett's Introductions to Plato.

This quotation formed the theme for Sir William Osler's lecture on the Educational Value of the Medical Society![1] I would like to quote several passages from that lecture:

"There are many problems and difficulties in the education of a medical student, but they are not more difficult than the question of the continuous education of the general practitioner. Over the one we have some control, over the other, none. The Universities and the state board make it certain that one has a minimum, at least, of professional knowledge, but who can be certain of the state of that knowledge of the other in five or ten years from the date of graduation?

"No class of men needs to call to mind more often the wise comment of Plato that education is a life-long business . . . The more serious problem relates to the education of the practitioner after he has left the schools. The foundations may not have been laid upon which to erect an intellectual structure, and too often the man starts with a total misconception of the prolonged struggle necessary to keep the education he has, to say nothing of bettering the instruction of the schools. As the practice of medicine is not a business and can never be one, the education of the heart—the moral side of the man—must keep pace with the education of the head . . . The doctor's post-graduate education comes from patients, from books and journals, and from societies, which should be supplemented every five or six years by a return to a post-graduate school to get rid of an almost inevitable slovenliness in methods of work."

Osler, in this lecture, was concerned primarily with the educational value of the medical society. Most specialty societies and national societies have continued with education of the members as the primary objective. The same cannot be said about many county medical societies.[2] In this group, affairs of the business of medicine are consuming a greater portion of the time and effort and matters of education of the members, a smaller portion.

Today, with this change in local medical societies, I am certain that Sir William Osler would suggest returning to a postgraduate school more often than once every five or six years. Indeed, most physicians in Oklahoma participate in postgraduate programs more frequently than this. A review of the records of the University of Oklahoma Postgraduate office is of some interest. During the year 1958-1959 there were 20 courses offered. The courses included Internal Medicine, Surgery, Pediatrics, Obstetrics, Orthopedics and many other subspecialties. Thirty-eight guest lecturers from other states served as instructors in addition to the local faculty. A total of 1,190 physicians attended one or more courses. The distribution of these physicians by residence is as follows:

Oklahoma County	429
Tulsa County	77
Other Oklahoma Counties	325
Out-of-State	309

One man attended 13 courses! A substantial number attended two or more courses. It would be anticipated that a large number of Oklahoma County physicians would attend, but the 375 attending from counties other than Oklahoma and Tulsa represents over 40 per cent of the practicing physicians. The growth of the program is indicated by the figures for 1949-50 compared with 1958-59.

	Physicians Attending Postgraduate Courses		
Year	Total Physicians	Out-of-State	Physicians
1949-50	211	14	
1952-53	247	27	
1958-59	1,190	309	

The figures for 1952-53 are included since at about this time the development of full-time teachers in the clinical departments had its beginnings and associated with this there was renewed interest in the continuing education of physicians.

The future development of the postgraduate program poses many questions.

The first is, should the postgraduate effort be one of continuing or reparative education? In other words, should the emphasis be on courses designed for the physician who has made a continued effort to educate himself, or should the courses be designed for the physician who has fallen shamefully behind in his education. The position of the American Medical Association Council on Medical Education and Hospitals is that the emphasis should be on continuing education.[3]

Should the subject be more basic or more practical?[2] Those that have advocated more practical to me, when queried as to what they meant by more practical, have invariably indicated more therapeutic. If a sound understanding of fundamentals is important and if concepts change as new methods become available, e.g., the development in understanding hemoglobinopathies with advent of electrophoretic techniques, then whose job is it to present these advances in fundamental knowledge? The University would seem to me to be remiss if it shirked this duty.

What method of presentation should be employed?[2] At the present, the lecture system is used almost exclusively. It is easier and cheaper but has few other advantages. Can seminars be incorporated into the program? The consecutive case conference has had wide application in the field of pulmonary disease.[4] In these conferences, each hospital participating presents 10 consecutive cases in some category, e.g., 10 consecutive cases admitted for tuberculosis. Usually two guest participants then criticize and discuss the cases. Could this method be applied for other subjects?

Where should courses be located?[2] The program of the University has been handicapped by an abominably poor auditorium with poor acoustics, uncomfortable seats and in general a rather barn-like atmosphere. Fortunately plans are underway to construct a new auditorium exclusively for the use of the postgraduate division. Should the courses all be given at the University of Oklahoma Medical School, or should some or all be given at other places in the State?[2,5] If so, where and how often? If this is done, can we induce the recalcitrant to drink in the educational pool, or will we still be satisfying only the thirsty?

How should the courses be financed?[2,5] If this is entirely by tuition, would physicians be willing to pay the cost or would attendance lag? Should the courses be financed by appropriated funds from the State Legislature? Should medical societies participate in the financing? Some do now, e.g., Oklahoma City Internists Association, Oklahoma Chapter of the American College of Surgeons, Oklahoma City Surgical Society, Oklahoma Association of Radiologists, Oklahoma Association of Pathologists, Oklahoma City Pediatrics Society, Oklahoma City Academy of Ophthalmology and Otolaryngology, Oklahoma Obstetrical and Gynecological Society, and others. Should county medical societies and the State Medical Association help support the program at the University? Should the volunteer health agencies help support courses for physicians? The Oklahoma Heart Association, Oklahoma Tuberculosis Association, American Cancer Society and others do this now. Should pharmaceutical firms help support the program as they have in the past?

The answers to all of these questions require thoughtful consideration and subsequent experimentation.[2] Many will remain only partially answered. Your suggestions and criticisms are welcomed by the postgraduate office. I would submit that your criticism should be based on evidence. Recently a physician was critical of the postgraduate program at the University. On inquiry it developed that he had not attended a course in six years!

The University and the physicians in the State, working together, can develop a program that will serve the needs for continuing education of physicians.—*J.F.H.*

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Fluid, Electrolyte and Nutritional Balance

The
First Oklahoma Colloquy
on
Advances in Medicine

Edited by

JAMES F. HAMMARSTEN, M.D.
Associate Professor of Medicine
University of Oklahoma School of Medicine and
Chief, Medical Service
Veterans Administration Hospital
Oklahoma City, Oklahoma

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Introduction

Stewart G. Wolf, Jr., M.D.

FOR THE FIRST Oklahoma Colloquy we have selected a topic which is at once intricate and broad. It is one in which the large amount of current experimental activity has not been adequately interpreted to the medical public. Since the concepts are difficult to grasp for those with a medical school background of fifteen or more years ago, we have selected the essayists for their articulateness as well as their stature in the field. With the aid of this distinguished group, we are attempting to make a badly needed contribution to post graduate education instead of a didactic review of the subject at hand. The men who are doing the work have presented their data in a comprehensive way but without attempting to cover the field. The questions and discussions from the audience and vigorous interchanges among the essayists themselves which are stimulated have been recorded. The flavor was that of a research meeting without its esoteric, fragmentary and highly specialized character. We are grateful to Baxter Laboratories for providing the financial support that made this Colloquy possible, and to the *Journal* of the Oklahoma State Medical Association for its publication and especially for making the entire Colloquy available for reference in a single issue of the *Journal*.

Table of Contents

The Renal Regulation of Acid-Base Balance: A Summary of Current Views, *Arnold S. Relman, M.D.* and *William B. Schwartz, M.D.* 265

The Relations of Potassium Depletion to Renal Structure and Function, *William B. Schwartz, M.D.* and *Arnold S. Relman, M.D.* 273

Aldosterone and Potassium Metabolism, *Robert E. Cooke, M.D.* 277

Acute Renal Insufficiency, *Ben I. Heller, M.D.* 282

Electrolyte Alterations in Surgical Patients, *William E. Abbott, M.D., Harvey Krieger, M.D.* and *Stanley Levey, Ph.D.* 290

A Practical Guide to Fluid Therapy in Burns, *Curtis P. Artz, M.D.* 296

The Practice of Fluid Therapy, *Robert Tarail, M.D.* 303

Use of Osmotic Diuretics in Surgery, *J. U. Schlegel, M.D.* 308

The Problem of Water Intoxication in Pediatrics and Surgery, *Robert E. Cooke, M.D.* 314

Central Nervous System Factors in Water and Electrolyte Metabolism, *John H. Bland, M.D.* 319

Stress: Its Effect Upon Fluid and Electrolyte Metabolism, *William W. Schottstaedt, M.D.* and *Robert Barnes, M.D.* 327

Role of the Connective Tissue System in Water and Electrolyte Metabolism, *John H. Bland, M.D.* 333

The Metabolic Response to Injury, *Curtis P. Artz, M.D.* 342

Clinical Aspects of Magnesium Metabolism, *James F. Hammarsten, M.D.* and *William O. Smith, M.D.* 354

Magnesium and Calcium Interrelationships, *Leonard P. Eliel, M.D., William O. Smith, M.D.* and *Carl Thomsen, M.D.* 359

Clinical Studies in Patients Receiving Long-Term Infusions of a Fat Emulsion, *John F. Mueller, M.D.* and *Fernando E. Viteri, M.D.* 367

Physiological Observations on Fat Transport Utilizing an Intravenous Emulsion, *John F. Mueller, M.D.* and *Jack Iacono* 373

The Use of Fat Emulsion, Alcohol and Protein Hydrolysates in Surgical Patients, *William E. Abbott, M.D., Harvey Krieger, M.D.* and *Stanley Levey, Ph.D.* 385

Participants

ARNOLD S. RELMAN, M.D.—Associate Professor of Medicine, Boston University School of Medicine, Boston, Massachusetts

ROBERT E. COOKE, M.D.—Professor of Pediatrics, Johns Hopkins School of Medicine, Baltimore, Maryland

BEN I. HELLER, M.D.—Professor of Medicine, University of Arkansas, Little Rock, Arkansas

WILLIAM E. ABBOTT, M.D.—Associate Professor of Surgery, Western Reserve University, Cleveland, Ohio

CURTIS P. ARTZ, M.D.—Associate Professor of Surgery, University of Mississippi, Jackson, Mississippi

ROBERT TARAIL, M.D.—Metabolic Section, Roswell Park Memorial Institute, Buffalo, New York

J. U. SCHLEGEL, M.D.—Director, Urological Research Laboratory, Assistant Professor, Urological Surgery, University of Rochester, Rochester, New York. Presently Professor of Urology, Head of Division of Urology, Department of Surgery, Tulane University School of Medicine, New Orleans, Louisiana

JOHN H. BLAND, M.D., Associate Professor of Medicine, University of Vermont Medical School, Burlington, Vermont

WILLIAM W. SCHOTTSTAEDT, M.D.—Associate Professor of Medicine and of Preventive Medicine and Public Health, University of Oklahoma School of Medicine, Oklahoma City, Oklahoma

JAMES F. HAMMARSTEN, M.D.—Chief, Medical Service, Veterans Administration Hospital, Associate Professor of Medicine, University of Oklahoma School of Medicine, Oklahoma City, Oklahoma

WILLIAM O. SMITH, M.D.—Assistant Chief, Radioisotope Service, Veterans Administration Hospital, Assistant Professor of Medicine, University of Oklahoma School of Medicine, Oklahoma City, Oklahoma

LEONARD P. ELIEL, M.D.—Professor of Research Medicine, University of Oklahoma School of Medicine, Director of Research and Head of Cancer Section, Oklahoma Medical Research Foundation, Oklahoma City, Oklahoma

JOHN F. MUELLER, M.D.—Assistant Professor of Medicine, University of Cincinnati, Cincinnati, Ohio

The Renal Regulation of Acid-Base Balance: A Summary of Current Views

ARNOLD S. RELMAN
WILLIAM B. SCHWARTZ

CONTINUOUSLY, in the course of health and disease, the extracellular fluid is being assaulted by acids and bases of exogenous and endogenous origin. Despite this, even in the most extreme acid-base disturbances, blood pH rarely varies by more than 3 or 4 tenths of a pH unit. This fluid owes its remarkably stable hydrogen ion concentration in part to the buffering actions of the carbonic acid-bicarbonate system and the numerous other buffer systems in red cells, bone and soft tissues with which the extracellular fluid appears to be in equilibrium. The lungs also contribute their vital share to the acid-base defenses by providing a route for the regulated excretion of CO_2 from the blood. Excretion of acid (hydrogen) or alkali (bicarbonate or other titratable alkaline anions) is, however, a function reserved exclusively to the kidney. It is only because of this renal activity that the body buffers can be restored to their original equilibrium state after the initial impact of the acid or base load has been absorbed. Without renal function, buffering capacity would quickly be exhausted and the pH of the body fluids would be at the mercy of whatever excess acids or bases chanced to appear.

Renal regulation of acid-base balance is primarily a tubular activity since the glomerular function of the kidney appears to be simply an ultra-filtering step which is inherently incapable of changing the acidity of the plasma. Three operations of the tubule can be distinguished in this regard: A) Reabsorption of bicarbonate, B) Excretion of acid buffered as ammonium, monobasic phosphate and other stronger acids, and C) Excretion of free hydrogen ion in minute quantities, i.e., establishment of a urine to plasma hydrogen ion gradient. Very little is known about the molecular basis of these operations, hence it cannot be stated to what extent they represent distinct or interrelated mechanisms. The distinctions made here are purely for descriptive purposes, although, as will be made clear, they are not without a certain amount of experimental support.

A. *Excretion and Reabsorption of Bicarbonate.* The bicarbonate ion is freely filtered at the glomerulus and distributed across the glomerular membrane according to the Gibbs-Donnan equilibrium. Considering the relatively huge daily volume of glomerular filtrate (180 liters) this means that a quantity of bicarbonate many times larger than the amount in extracellular fluid is poured into the renal tubules each day. One of the important factors determining the concentration of bicarbonate in the extracellular fluid is therefore clearly the rate at which this ion or some product thereof is reabsorbed by the renal tubules.

Micropuncture studies in amphibia and mammals provide evidence that under normal circumstances bicarbonate is removed in proportion to water all along the proximal convoluted tubules. At the end of this portion of the nephron approximately 85% of the glomerular filtrate and its contained bicarbonate have disappeared, and the pH of the tubular fluid is still that of the plasma and the glomerular filtrate. However, virtually none of the filtered bicarbonate escapes reabsorption, because practically all that enters the distal tubule is removed by the time the final composition of the urine has been achieved. It is during this final reabosorptive process that the pH of the tubular fluid is reduced to that observed in the urine. Whether the reabsorption of *all* bicarbonate, including the most distal fraction, is accomplished by a single mechanism cannot be stated at present.

On the basis of a good deal of circumstantial evidence which cannot be detailed here, it is widely believed that most, if not all, bicarbonate is removed from the filtrate by tubular secretion of hydrogen ions and conversion of the filtered bicarbonate to carbonic acid. It is thought that this reaction is accomplished by an ion exchange process in which hydrogen ions from the tubular cell are exchanged for sodium ions in the glomerular filtrate. In the tubular fluid carbonic acid then disassociates into CO_2 and water, and the highly diffusible CO_2 rapidly moves back into the

plasma. In the process of secreting hydrogen ions the tubular cell simultaneously generates alkali which diffuses out of the cell into the plasma in the form of bicarbonate. Thus, the bicarbonate ion which appears in the renal venous plasma as the net result of the reabsorptive process is not actually the same ion which was removed from the filtrate, but the total effect is as if this ion were directly taken from the filtrate back into the blood. It seems likely that carbonic anhydrase activity inside tubular cells is necessary for reabsorption of bicarbonate since this process can be immediately slowed by the administration of a fairly specific enzyme inhibitor such as acetazolamide. The function of carbonic anhydrase is believed to be in catalyzing the reactions

$$1)\ CO_2 + H_2O \rightleftharpoons H_2CO_3$$

$$\text{and } 2)\ CO_2 + OH^- \rightleftharpoons HOC_3^-$$

Reaction 1 proceeding rapidly in the direction left to right might provide the source of intracellular hydrogen ions necessary for H-Na exchange. Alternatively, the hydrogen ions might come from other intracellular acid sources and the essential function of the enzyme might consist in facilitating the shift of Reaction 2 from left to right, allowing CO_2 to buffer the hydroxyl ions which would otherwise be accumulating in the tubular cells as they continued to secrete hydrogen ions.

For practical purposes, these two possible functions of carbonic anhydrase would be indistinguishable. In either scheme, a carbonic anhydrase-catalyzed reaction would be an essential step in the reabsorptive process and a critical reduction in enzyme activity would be expected to lower the renal threshold for bicarbonate. Carbonic anhydrase appears to be involved in the reabsorption of at least the great bulk of the filtered bicarbonate, since administration of acetazolamide under normal circumstances may divert more than half of the filtered load into the urine. Even when the plasma bicarbonate level is very low and the initial rate of reabsorption a small fraction of the normal, inhibition of carbonic anhydrase will appreciably diminish the rate of reabsorption.

Under normal circumstances virtually all of the filtered bicarbonate is reabsorbed when the plasma bicarbonate level is substantially below normal. As the plasma level is progressively raised by infusion of bicarbonate, increasingly large amounts of bicarbonate appear in the urine beginning at plasma levels around 24 or 25 mEq/L. Net reabsorption of bicarbonate, calculated as the difference between the filtered load (plasma level x glomerular filtration rate) and the rate of excretion, remains constant despite progressive elevation of plasma level. This abrupt saturation of the capacity of the tubule to reabsorb bicarbonate establishes a fairly sharp renal threshold for this ion and thereby determines the upper limit of the plasma bicarbonate concentration. Only by continuous and rapid administration or endogenous generation of alkali could the blood bicarbonate level be maintained above the renal threshold for any length of time.

Changes in renal threshold occur in a variety of clinical and physiological circumstances and are of the greatest importance in understanding acid-base adjustments in health and disease. A partial list of the factors which influence bicarbonate reabsorption include: 1) the CO_2 tension of the blood (pCO_2), 2) potassium balance, 3) sodium balance, and 4) drugs (e.g., acetazolamide) and 5) tubular diseases which may temporarily or permanently interfere with the integrity of the reabsorptive mechanism.

It has been found that the renal bicarbonate threshold tends to vary directly with the blood carbon dioxide tension. Precisely how this is accomplished is not known, but the physiological and clinical importance of this relationship is obvious. In respiratory acidosis the blood bicarbonate level tends to rise as an initial result of the buffering of carbonic acid by the hemoglobin or red blood cells and probably by buffers in other tissues as well. Later, increased renal excretion of ammonium and possibly also titratable acid serve further to raise the bicarbonate, as will be discussed in a subsequent section. The rise in blood bicarbon-

ate, tends to neutralize the acidifying effect of the increased pCO_2 and is essential to the maintenance of a normal blood pH. If the renal threshold for bicarbonate did not rise under these circumstances the extra bicarbonate in the blood would immediately spill out into the urine and effective compensation would be impossible. Actually, however, chronic elevation of blood CO_2 tension results in an appropriate rise in bicarbonate threshold which keeps the bicarbonate in the blood.

The reverse situation is seen in respiratory alkalosis. Sudden reductions in blood CO_2 tension resulting from increases in ventilatory volume result in a shift of bicarbonate ions into red cells and perhaps other tissues in exchange for chloride. The resultant drop in blood bicarbonate tends to minimize the rise in blood pH resulting from the reduced CO_2 tension, but were it not for a sudden increase in the renal excretion of bicarbonate, extreme rises in pH would nevertheless occur. Diuresis of bicarbonate in this situation is the result of a reduction in the tubular reabsorption attendant upon the change in pCO_2.

This physiological relation between respiratory function and bicarbonate reabsorption may not always serve the best interests of acid-base equilibrium. In chronic metabolic alkalosis the blood bicarbonate level is elevated due to excessive ingestion or endogenous production of alkali, or to excessive loss of acid from the body. Respiratory compensation for the rise in blood pH may sometimes result in appreciable reductions in ventilation and some rise in blood pCO_2. This, in turn, elevates the renal bicarbonate threshold and therefore maintains the abnormally high blood bicarbonate levels, even after the initiating cause of the alkalosis may have disappeared.

Changes in the tubular transport of either sodium or potassium may have important effects on the renal reabsorption of bicarbonate. Since the first step in the reabsorptive process appears to be an exchange of sodium in the filtrate for hydrogen in the tubular cells *(vide supra)* it would seem reasonable to expect that circumstances which stimulate sodium reabsorption would also tend to favor bicarbonate reabsorption and thus to increase the renal threshold for bicarbonate. Stimulation of sodium reabsorption by sodium depletion or by administration of adrenocortical salt retaining hormones has been found to have precisely this effect, although the action of the latter probably involves loss of potassium as well.

The relationship between potassium metabolism and bicarbonate excretion seems best explained at present by the hypothesis that potassium is excreted to a large extent, if not entirely, by tubular secretion, and that potassium and hydrogen ions compete for the same secretory pathway. Secretion of both ions is thought to involve the simultaneous reabsorption of sodium. According to this view, potassium-deficiency and a lowered concentration of potassium in tubular cells would favor exchange of hydrogen for sodium and would tend to increase the reabsorption of bicarbonate. Conversely, increased amounts of potassium in the blood and in the renal tubular cells forces increased secretion of this ion and competitively inhibits the simultaneous secretion of hydrogen. This would tend to reduce the rate of bicarbonate reabsorption and tend to alkalinize the urine.

Many clinical and experimental facts appear to fit this hypothesis. There is a high renal threshold for bicarbonate in states of potassium-deficiency, and this is largely responsible for the maintenance of the high blood bicarbonate levels which are such a frequent feature of potassium-depletion. (The kidney, however, is not usually the *cause* of this alkalosis, which apears in many cases to be due to a shift of extracellular hydrogen into cells as potassium is lost.) Despite the increased bicarbonate threshold in this situation, the urine pH tends to rise slightly as the potassium-deficiency develops, indicating a decrease rather than increase in the bicarbonate reabsorbed at some distal point in the tubule.

An elevated blood pCO_2 may also play a role in increasing bicarbonate reabsorption in states of potassium-deficiency, but it has been shown that the rise in threshold at any

given elevation of pCO_2 is greater in the presence of potassium-depletion. The converse relationship between potassium and bicarbonate can also be demonstrated. Rapid infusion of potassium in normal subjects will abruptly lower the threshold, increase bicarbonate excretion and produce metabolic acidosis. Even when the threshold is elevated by a high pCO_2, infusion of potassium will cause a bicarbonate diuresis.

Changes in carbonic anhydrase activity are also responsible for important effects on bicarbonate excretion. The acidosis produced by administration of acetazolamide is thought to be the direct result of inhibition of tubular carbonic anhydrase and the consequent reduction in bicarbonate reabsorption. Immediately following the administration of this drug there is a bicarbonate diuresis and the blood bicarbonate level falls. Continued administration of the drug results in stabilization of the blood bicarbonate at some moderately low level, but the urine bicarbonate excretion tends to return to pretreatment levels. This phenomenon also accounts for the self-limiting diuretic action of this compound, and its ineffectiveness in patients with metabolic acidosis. In acute experiments in dogs doses equivalent to those used clinically have little or no effect when they are administered at plasma bicarbonate levels below 12 to 15 mEq/L.

In renal disease acidosis results from tubular damage and impairment of some or all of the mechanisms for maintenance of acid-base balance. In some cases the renal threshold for bicarbonate has been found to be significantly reduced. It is possible that this is due to a reduction in carbonic anhydrase in the tubular cells, although other explanations can not presently be ruled out. In such patients elevations of blood bicarbonate to normal levels by infusion of bicarbonate results in rapid excretion of this ion until the plasma concentration has fallen to the level of the renal threshold. At this point bicarbonate excretion in the urine ceases to be significant and in some patients a further degree of acidosis may even stimulate the production of urines of pH 5.0-5.5. Patients with so-called renal tubular acidosis have more difficulty in acidifying their urine, the pH of which is usually found to be above 6.0 even with severe acidosis. However, when they are loaded with bicarbonate, the magnitude of their urinary bicarbonate "leak" is no greater than that of some patients with other forms of renal disease who excrete more acid urines when they are acidotic.

B. *Excretion of Acid.* The two important mechanisms by which the kidney gets rid of acid from the body are: 1) excretion of hydrogen buffered in the form of weak acids ("titratable acidity"), and 2) excretion of hydrogen buffered by ammonia in the form of ammonium ion. The net effect of either process on acid-base economy is the same, since a milliequivalent of hydrogen is lost from the body with each milliequivalent of ammonium or titratable acid. This means that the plasma returning from the kidney must have a higher concentration of bicarbonate than that in the renal artery because removal of hydrogen has the same chemical effect as addition of alkali.

Normally, the most important weak acid excreted in the urine is the monobasic phosphate ion ($H_2PO_4^-$, pK′ approximately 6.8). To a much smaller extent, hydrogen is also carried out in the form of relatively weak organic acids such as creatinine (pK′ approximately 4.97). Since the pH of the urine rarely gets much below 4.5, it is clear that the concentration of hydrogen ions excreted in the urine is always very low (less than 10^{-4} M/L) and almost no titratable hydrogen can be excreted in the form of acids with pK's below 3.0. The term "titratable acid" ordinarily refers to the urinary hydrogen which can be titrated between the pH of the urine and that of the blood. This virtually excludes all acids with very low pK's, e.g., the mineral acids and the stronger organic acids or very high pK's (e.g., NH_4^+).

Conversion of alkaline to acid phosphate ($HPO_4^- \rightarrow H_2PO_4^-$) is thought to be accomplished in the distal part of the convoluted system by the same kind of sodium-hydrogen exchange mechanism as that involved in the reabsorption of bicarbonate. It cannot be said for certain that this process, like that for bicarbonate, is dependent on carbonic

anhydrase activity, although this seems likely. Administration of acetazolamide increases excretion of bicarbonate and therefore raises the pH of the distal tubular fluid. Even if the secretion of hydrogen for excretion as titratable acid were a process separate from bicarbonate reabsorption and were not directly inhibited, the net effect of alkalinization of the urine would be to reduce the excretion of phosphate in the acid form.

On ordinary diets under usual conditions normal subjects excrete about 20 or 30 milliequivalents of titratable acid per day, but under certain circumstances this rate may be substantially reduced or may be increased by several fold. The maximal excretion of titratable acid is determined by the urine pH and by the total amount of buffer that can be excreted. Titratable acid may be greatly increased by infusion of phosphate into acidotic subjects, but unless there is a disturbance such as diabetic acidosis which leads to an increased excretion of one or more organic acids, there is ordinarily only a modest increase in titratable acid excretion in acidotic states. This results from the fact that in metabolic acidosis, for example following ammonium chloride administration, there is only a slight phosphate diuresis. Since the pK′ for monobasic phosphate in urine is approximately 6.8, it is clear that relatively little increase will be produced by reducing urine pH much below 6.0. In contrast, in diabetic acidosis where there is a large increase in the excretion of diacetate (pK′ approximately 3.8) and betahydroxy butyrate (pK′ approximately 4.7), titratable acid excretion may rise to more than 100-150 mEq/day. In this latter circumstance the pH of the urine is of great importance since the maximum buffering of these compounds in urine takes place below a pH of 5.5. Most patients with acidosis due to renal disease, except some with so-called renal tubular acidosis, are able when acidotic to elaborate urines of pH 6.0 or below, and hence they may excrete approximately normal amounts of titratable acid. However, if they have a low bicarbonate threshold, as noted above, their urines may be quite alkaline when attempts are made to raise their plasma bicarbonate toward normal, and under these circumstances they can excrete little or no titratable acid.

Usually, the greatest burden of urinary hydrogen is carried by the ammonium ion. In states of severe chronic acidosis healthy kidneys may excrete as much as 300-500 mEq/day. No direct information exists concerning the exact mode of excretion of ammonium. It is known that ammonia is synthesized in the renal tubules by enzymatic deamination of alpha-amino acids, chiefly glutamine. Glutaminase activity and the availability of amino acid substrate are therefore important factors in determing the production of ammonia. Because ammonia is a relatively strong base (pK′ approximately 9.3) and the maximum urinary pH is rarely above 8.0, almost all must be excreted in the acid form as the ammonium ion. All other things being equal, the urinary excretion of ammonium seems to be an inverse function of the urine pH, and this is also true of the excretion of certain other organic bases which are believed to be secreted by the tubules and have pK values similar to ammonia. For this reason, and also because cell membranes are usually more permeable to the free ammonia base than to the ammonium ion, it is now believed that it is the free base which diffuses into the tubular fluid. Hydrogen transferred to the tubular fluid in the process of sodium reabsorption is then buffered by combination with the base to form ammonium. The dependence of ammonium excretion on urine pH may be explained by the greater tendency of the free base to diffuse into an acid medium than an alkaline one. As the pH of the tubular fluid rises, the concentration of the free base also rises and hence the gradient for diffusion from the tubular cells into the fluid would fall.

The physiological factors regulating excretion of ammonium are not well understood. Ammonium excretion in various clinical and physiological states is clearly not simply a passive function of the urine pH. It is often found to vary independently of the latter. Furthermore, the correlation with blood pH is rather poor, although the acidity of renal tubular cells may be more directly concerned with ammonium excre-

tion. With continuous administration of acidifying salts to normal subjects, ammonium excretion rises progressively until it is virtually equal to the administered acid load. This rise occurs despite the fact that after an initial fall urine and blood pH remain fairly constant. It has been shown that prolonged acidosis causes some sort of enzymatic adaptation, with an increase in glutaminase activity and a greater capacity for tubular production of ammonia.

Another important factor which appears to influence ammonium excretion and which may play a role in the increasing response to continued loading with acid salts is the intensity of the stimulus to sodium reabsorption in excess of urinary anion. The ammonium response to a given load of ammonium chloride is greater in salt-depleted subjects, and salt-depletion becomes progressively more severe as the ammonium chloride is administered. Administration of DOCA causes an immediate increase in ammonium excretion, but only when there is a substantial amount of sodium in the diet. In neither situation can these changes in ammonium excretion be correlated with an increased acidity of urine or blood. A more reasonable explanation appears to reside in the sodium transport mechanism, since reabsorption of sodium without accompanying anion would tend to create a potential gradient which would favor the excretion of other cations, such as ammonium and potassium.

Potassium metabolism also influences ammonium excretion. When tubular cells are deficient in potassium as the result of serious body deficits of this ion, ammonium excretion tends to be somewhat higher than might be expected from the relatively high urine pH so characteristic of this condition. This effect may be due to a number of factors: a) renal glutaminase activity is increased in experimental potassium-depletion, b) the acidity of tubular cells may be increased, thereby favoring the H-Na exchange which is ultimately responsible for ammonium excretion, and c) less cellular potassium is available for secretion, thus increasing the electrostatic requirement for cation in the form of ammonium. The high ammonium excretion in primary aldosteronism is probably due in part to the severe coexisting deficiency of potassium. Furthermore, as might be predicted from the relationship of ammonium excretion to sodium reabsorption, excretion of ammonium in aldosteronism is increased when salt is added to the diet and reduced when salt is withdrawn.

In renal acidosis ammonium excretion is almost always reduced, even when urine pH is low. This is presumably due to structural or biochemical defects which sharply limit the capacity of the tubular cell to generate free ammonia or to a reduction of total tubular mass. In patients with constantly alkaline urines, such as those with so-called renal tubular acidosis, an unfavorable diffusion gradient for ammonia may also be a limiting factor. Whatever the explanation, the inadequate ammonia response in renal patients is clearly a major factor in the pathogenesis and maintenance of acidosis, usually of greater importance than any reduction in the capacity to excrete titratable acid. Without an ability to greatly increase the excretion of ammonium, patients with renal disease cannot effectively regenerate their body buffers and therefore are at the mercy of dietary or endogenous acids.

C. *Acidification of the Urine.* The glomerular filtrate is formed at the pH of the plasma, but bladder urine may be as alkaline as pH 8.0 or as acid at 4.0. Changes in pH are apparently accomplished in the distal convoluted tubules. Maximal acidification of the urine requires that almost all the bicarbonate remaining in the tubule be removed, and that any urinary buffers with pK's in the range of 3.5-5.0 be at least partly titrated with hydrogen. Although the pH of renal tubular cells is not known precisely, it may be estimated that at some distal point in the tubule hydrogen ion gradients across the cell membrane may approach 1000 to 1. It is assumed that this final operation involves the same sodium-hydrogen exchange process which is responsible for the reabsorption of bicarbonate, and the excretion of ammonium and titratable acid by the other parts of the distal tubule. However, there is some evidence to suggest that the acidifying function can vary independently of the

bicarbonate threshold and the capacity for ammonium excretion. In severe potassium-depletion, for example, there is a defect in acidification, but ammonium excretion and the threshold for bicarbonate are increased. In certain types of renal disease, furthermore, it has been observed that the capacity to acidify the urine may be relatively well preserved even when the bicarbonate threshold is lowered and ammonium excretion sharply curtailed.

Little is known about the physiological regulation of urine acidity. In dogs there is relatively little response to acute respiratory or metabolic acidosis even when very severe. In general, however, the urine pH does tend to vary with the acid-base balance of the body under many circumstances. There is some reason to believe that the acidification function and ammonium excretion bear the same relation to sodium reabsorption. It can be shown, for example, that rapid infusion of sodium sulfate will cause intense acidification of the urine if the subject has previously been stimulated to retain part of the infused sodium load by a low sodium diet and adrenal steroids. Under normal circumstances the same sulfate load is without effect on urine pH. It is possible to explain this phenomenon by postulating that acidification will occur whenever distal sodium reabsorption is rapidly increased in excess of anion and when the unreabsorbed anion remaining in the urine is of very low buffer capacity. An inportant stimulus for acidification may well prove to be sodium reabsorption rather than any direct or immediate consequence of acidosis.

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DISCUSSION

DOCTOR L. L. CONRAD, Assistant Professor of Medicine, University of Oklahoma School of Medicine, Chief, Cardiovascular Disease Section, Veterans Administration Hospital, Oklahoma City, Oklahoma: Doctor Relman, would you review those factors which are pertinent in the determination of intracellular pH? What are the determining factors, and how do you determine intracellular pH?

DOCTOR RELMAN: That is a very difficult question to answer. Most of our information about intracellular pH is indirect. The actual measurement of hydrogen ion concentration inside living cells is a formidable technical problem which has not been satisfactorily solved. We can measure the bicarbonate, or I should say we can measure the total CO_2 content of the tissues, and if one depletes tissues of potassium, the total CO_2 content goes down. If one keeps these tissues in a constant CO_2 tension, then making some

reasonable assumptions about the dissociation constant for carbonic acid inside tissues, one can say that pH has gone down; but the fact of the matter is, most of our information about intracellular pH is largely a matter of bookkeeping. You calculate with reasonable accuracy how much potassium has left the cell, you calculate with somewhat less accuracy how much sodium has gone into the cell, and then you say somewhat hesitantly with not very much confidence that maybe the discrepancy between the potassium out and the sodium in is accounted for by hydrogen ion. This is rather uncertain, and we have a lot to learn about intracellular pH.

DOCTOR CONRAD: I have a question for Doctor Heller or Doctor Relman. Is plasma bicarbonate content or plasma pH any index of tissue potassium deficit in metabolic alkalosis with hypokalemia?

DOCTOR HELLER: I would say that tissue potassium deficits may be associated with elevations of plasma bicarbonate content, and thus with increase in the pH, but all states in which there is an increase of the plasma bicarbonate content need not necessarily be associated with potassium deficit. In general, when hypokalemia and metabolic alkalosis co-exist, I think that the degree of metabolic alkalosis is roughly correlated with the degree of tissue potassium deficit. Potassium depletion, however, may be associated with a high pH, a low pH, or a normal pH.

DOCTOR RELMAN: I would like to add one comment. I think that the fairly quantitative relationship between extracellular bicarbonate and intracellular potassium holds true for very restricted and specific experimental situations, namely the type of potassium depletion produced, but in clinical practice one can see potassium deficiency occurring with acidosis or with alkalosis or with a normal pH. The type of acid-base disturbance and the severity of this disturbance in clinical practice, as far as I can see, has no predictable relationship whatsoever with intracellular potassium deficit. Thus, for example, in uremia you can have potassium depletion with acidosis; and in severe diarrhea, with loss of alkali in the stools, you can have potassium depletion with acidosis. With ammonium chloride poisoning, you can have acidosis and potassium depletion.

So, I would certainly agree with Doctor Heller and I would emphasize his point that it depends on how the potassium depletion is produced.

DOCTOR CONRAD: What is your experience with Diamox in respiratory acidosis with chronic pulmonary disease? Would you care to comment on that?

DOCTOR RELMAN: Doctor Leaf, Doctor Schwartz and I[1] a number of years ago treated a series of patients with respiratory acidosis and congestive failure with Diamox and observed some improvement with diuresis in about half of the group. We have not had any experience with pulmonary insufficiency or respiratory acidosis without congestive failure.

DOCTOR CONRAD: Patients with liver disease treated with Diamox may develop liver failure and elevated blood ammonia concentrations. Would you comment on that observation.

DOCTOR RELMAN: This is an interesting phenomena which has ben observed by a number of workers. Roberts and co-workers[2,3] at Memorial Hospital, have pointed out that the symptoms of ammonium intoxication are probably due to intracellular concentrations of free ammonia base. If I recall their work correctly, they produced symptoms of ammonium intoxication by respiratory alkalosis in compensated patients with potassium insufficiency and improved the symptoms with respiratory acidosis. And, of course, these two conditions would change intracellular pH.

Now it seems to me, although this is all speculation, that a reasonable hypothesis for the action of Diamox would be that by inhibiting carbonic anhydrase in some critical cells, perhaps the brain or elsewhere, changes intracellular pH, and it is this that may produce ammonium intoxication.

DOCTOR CONRAD: Would you compare the action of chlorothiazide with that of Diamox on the kidney tubule.

DOCTOR RELMAN: Chlorothiazide is a sulfonamide and does have some carbonic anhydrase inhibiting activity. Compared to Acetazolamide, or Diamox, it is relatively weak, but it will alkalinize the urine slightly and increase the excretion of bicarbonate slightly. It does not owe its diuretic action, however, to this effect, in contrast to Diamox, which usually produces no increase of chloride and increases sodium excretion only by increasing bicarbonate. Chlorothiazide does have a large effect on the reabsorption of sodium chloride, so that one does observe with the administration of Chlorothiazide a slight increase in the excretion of bicarbonate but a much greater increase in the excretion of chloride and sodium. Whether chlorothiazide works on the reabsorption of chloride primarily or on reabsorption of sodium, and whether it affects the same mechanisms that are affected by mercurial diuretics is, so far as I am aware, as yet unknown.

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The Relations of Potassium Depletion to Renal Structure and Function

WILLIAM B. SCHWARTZ
ARNOLD S. RELMAN

POTASSIUM IS an essential, although not entirely irreplaceable, structural component of all cells. It appears, furthermore, to be an activator of certain enzymatic reactions involved in intermediary metabolism. In renal tubular cells, however, potassium plays still another role. There, it is an active participant in the ion exchange reactions through which renal regulation of electrolyte and acid-base balance is accomplished. It is not surprising, therefore, that severe chronic depletion of potassium should have profound consequences for both renal structure and function.

Rats placed on potassium-deficient diets within a few days begin to develop renal enlargement, which is followed shortly by the appearance of swelling, hyperplasia and granular changes in the epithelium of the collecting tubules. Obstruction leads to dilation of the convoluted system proximally, and low-power examination of the kidney usually discloses a striking cyst-like band of dilated tubules at the cortico-medullary junction. More advanced changes include various types of degenerative and necrotic lesions of the convoluted tubular epithelium, but for the most part there is relatively little damage to glomeruli, blood vessels, or other interstitial structures.

Restoration of potassium to the diet can produce complete healing of the renal lesions, but in some instances interstitial scarring and hyalinization of glomeruli have been noted long after correction of severe chronic potassium-deficiency. This may mean that the lesions of experimental potassium-depletion can become irreversible at some point, although this question has not been definitely settled.

The striking histologic effects of potassium-depletion in the kidney are accompanied by significant changes in the composition of renal tissue. Potassium content of the kidney is usually reduced by about 10-20%, although it is not proportionately as low as in muscle tissue and is not associated with a demonstrable increase in sodium. Lysine and arginine are increased, as in potassium-depleted muscle. Carbonic anhydrase and glutaminase activity are also increased. Histo-chemical techniques reveal increased acid phosphatase, TPN diaphorase and non-specific esterase activity in the tubular epithelium.

In man the renal lesion of potassium-deficiency resembles closely that of the rat in being confined chiefly to the tubular epithelium. Unlike the murine lesion, however, the human nephropathy is primarily a vacuolar and hydropic degeneration of the convoluted tubules, with the collecting tubules remaining unchanged in appearance. The vacuoles may be quite striking in their number, size and discreetness, sometimes appearing to occupy almost all of the tubular cytoplasm. They do not stain for fat, glycogen or protein, and usually seem to be relatively more numerous in the proximal than in the distal tubules. In many cases, however, these distinctive changes are not present or are relatively sparse. Less specific changes in the tubular epithelium may occur, including granularity, foamy swelling of cytoplasm and occasionally necrosis or sloughing of cells. Glomeruli, blood vessels and interstitial tissue are usually normal, unless there happens to be some other coexisting pathological process. Virtually no precise information is avalable concerning the minimum duration and severity of the potassium-deficiency required to produce significant lesions. By history, most of the cases observed to date appear to have been losing potassium for at least a month and usually much longer. In a few instances studied by serial biopsy well-developed lesions have been shown to be completely reversible when the potassium-depletion was repaired, but the question of possible irreversible or progressive renal disease in man has not been settled.

Lesions characteristic of potassium-deficiency have been observed in a great variety of clinical disorders. The most common cause is severe chronic diarrhea of any etiology, but hyperadrenalism (including Cushing's disease and primary aldosteronism) as well as renal tubular disorders with

urinary wasting of potassium have also frequently been at fault. The lesions due to potassium-deficiency are not infrequently found together with other independent renal pathology. It has been suggested that potassium-depletion makes the kidney more susceptible to pyelonephritis. While pyelonephritis has been often found associated with aldosteronism, the incidence in other types of potassium-depletion does not appear strikingly high. This question needs further study.

The most characteristic functional defect associated with potassium-depletion in both man and rat is an impairment of concentrating ability. In rats, and occasionally in man, this is accompanied by polydipsia and polyuria. Other tubular functions have not been systematically studied, but fragmentary observations in patients suggest that tubular transport of PAH and phenol red may sometimes be impaired. Despite the absence of significant glomerular lesions, filtration rate has been found moderately reduced in patients who become depleted from diarrhea, and there is a suggestion that this may be true for experimentally depleted rats as well. Blood nitrogen levels may be slightly increased under these circumstances. It is of interest, however, that a number of patients with potassium-depletion nephropathy resulting from aldosteronism have had entirely normal filtration rates. In many instances all the functional defects have proven to be largely or entirely reversible within a few weeks or months after correction of the potassium deficits.

Because active secretion of potassium appears to be closely linked to the other tubular exchange processes involved in the reabsorption of sodium and the secretion of hydrogen ions, it is not surprising that the renal tubular regulation of electrolyte and acid-base balance is significantly affected in states of severe potassium-deficiency. Presumably, this is the direct result of reduced potassium content of tubular cells, but since there is reason to believe that cells deficient in potassium also have an increased sodium content and acidity, it is possible that these latter changes also affect the ion exchanges in the tubule.

The important changes with severe chronic potassium-deficiency appear to be the following:

1. When potassium-depletion has been caused by extrarenal losses, there is a marked reduction in urinary excretion of potassium. This is presumably due to reduced distal tubular secretion of potassium, since most of the filtered potassium is thought to be reabsorbed in the proximal tubule. When the deficiency is due to hyperadrenalism (e.g., Cushing's syndrome or primary aldosteronism) or potassium-losing renal disease, the urine potassium content will naturally be high. Excretion of less than 10 milliequivalents of potassium per day ordinarily means that there is at least a moderately severe body deficiency and that this has come about through extrarenal losses.

2. There is an increased capacity for bicarbonate reabsorption, resulting in a high renal threshold for this ion. This change is responsible for the maintenance of the elevated plasma bicarbonate levels which are a common, although not invariable, feature of posassium depletion.

3. There is a tendency toward increased sodium reabsorption, which is responsible for the occasional development of edema. In primary aldosteronism there is usually no edema, possibly because the glomerular filtration rate tends to be relatively higher than in most other states of postassium-deficiency.

4. Ammonium excretion is somewhat increased, despite the fact that the urine is slightly more alkaline than usual.

5. The urine pH in severe potassium-deficiency rarely falls below 6.0 spontaneously, and tends to have a slightly higher than normal content of bicarbonate.

A reasonable, although entirely tentative, interpretation of these observations is as follows: Decreased cellular potassium content is directly responsible for the reduced secretion and excretion of potassium and is also accompanied by a significant increase in cellular acidity. The latter, in turn, fa-

vors the secretion of hydrogen in exchange for sodium in the tubular fluid, simultaneously resulting in increased reabsorption of bicarbonate and sodium and increased excretion of ammonium. Increase in renal glutaminase activity caused by the change in cell composition may also increase ammonium excretion. The tendency toward relatively alkaline urine may be attributed to a non-specific distal tubular defect comparable to that which impairs the urine concentrating mechanism, since it is presumably in the distal tubule that the normal urine to plasma hydrogen ion gradient is established. Alternatively, the relatively high ammonium excretion may result in a tendency for the plasma bicarbonate to rise above the renal threshold, even though the latter be increased by the tubular deficiency of potassium.

All of the above functional disorders associated with potassium-deficiency can be fairly rapidly reversed by restoration of the normal potassium balance. Thus, it has been observed that administration of potassium to depleted subjects results in: a) gradual increase in potassium excretion, b) a large diuresis of sodium, c) an abrupt reduction in the threshold for bicarbonate, d) a gradual fall in urinary ammonium excretion, and e) a slow return of urine pH to the usual range.

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DISCUSSION

DOCTOR ELIEL: Your discussion of potassium deficiency, centered about acute and subacute clinical states, can the same result appear after long-standing substandard dietary intake of potassium?

DOCTOR RELMAN: I don't know. I don't see any reason why chronic potassium depletion should not do everything that acute and subacute depletion does; and in fact, most of the patients that we have studied who were acutely depleted were fairly chronically depleted over a period of many months or perhaps years.

DOCTOR ELIEL: In potassium deficient states, is there evidence that protein utilization is impaired in spite of good protein intake? And is potassium necessary for adequate protein and carbohydrate metabolism?

DOCTOR RELMAN: There are no data in humans that I am aware of, but the work of Doctor Paul Cannon, of the University of Chicago, and others is quite definite on this point with regard to experimental animals. Potassium depleted animals do not use protein normally.

Furthermore, protein depletion will modify many of the effects of potassium depletion. In animals, in order to get many of the biochemical and physiological disorders that we associate with potassium depletion, one has to start out with a well-nourished animal and deplete him of potassium.

DOCTOR ELIEL: Now the next series of questions deals with the tissue changes subsequent to potassium depletion. I think that I might pose all of them and ask Doctor Relman to answer first and then Doctor Cooke.

The question is what evidence is there that potassium depletion leads to lesions in other tissues, particularly rapidly growing tissues such as gastrointestinal epithelium and bone marrow?

Are these changes in renal physiology which you have described the result of abnormalities secondary to hypokalemia? In other words, there are many secondary changes which result from decreased potassium. What changes are due to deficit of potassium ion itself?

Finally, have there been any studies on potassium depletion in children with celiac disease? Doctor Relman.

DOCTOR RELMAN: The first question about lesions in tissues other than the kidney, as you mentioned, Doctor Eliel, there are often lesions in the myocardium. We have not studied these lesions. They have been studied in detail by many other workers, but focal lesions have been reported and I believe that focal lesions, degenerative lesions in the skeletal musecles, have also been described.

We have been chiefly interested in the renal lesions. Grossly, there are striking changes in the gastrointestinal tract of experimental animals. Rats dying with potassium depletion have gross distention of their intestinal tracts. Sometimes this is full of fluid and this reminds one, of course, of the paralytic ileus and the distention that is often seen in patients who are clinically depleted of potassium.

With regard to the second question concerning the point about whether it is potassium itself or some secondary effect of the potassium which causes the renal lesion, I certainly cannot say. It seems to me impossible at this time to answer that kind of question because we simply do not know enough about

exactly what potassium does inside of the cytoplasm. All that we can say is that if you take potassium away, you produce the lesion; if you add the potassium, it goes away. What the sequence of events is, at the molecular level which explains these lesions, I simply do not know.

DOCTOR ELIEL: Doctor Cooke, can you answer the question about the celiac disease?

DOCTOR COOKE: I would like first to add a little to what Doctor Relman said. The stomach was shown by one of my students[1] a few years ago to have marked changes in its capacity to excrete acid during potassium deficiency. We did not see any anatomical changes and I don't know what the significance of that is.

The effect on the bone is one of the most interesting; not from a pathological standpoint, but from a functional standpoint. This is referrable to studies of Finberg[2] in Baltimore on patients with hypernatremia who, with gradual lowering of the sodium concentration, developel marked hypocalcemia.

In rats, as you remember, if one raises the sodium concentration and then lowers this with glucose and water, there is a very marked drop in calcium concentration of extracellular fluid. If you put potassium in during this period of time, for some reason, calcium remains in the extracellular phase and hypocalcemia does not occur; so that I think the bone gets into this problem of potassium depletion, but exactly how, we don't know.

And as regards the celiac disease, I am personally not too well acquainted with the problem of potassium deficiency in celiac disease. Actually, celiac disease is a disappearing disease; and over the period in which there has been a lot of interest in potassium metabolism, there has been less celiac disease than there used to be.

As far as the cystic fibrosis of the pancreas is concerned, there is a report in the New England Journal[3] that came from the Hospital of Medical Evangelists in California on potassium deficiency in cystic fibrosis of the pancreas. These authors attributed the potassium deficiency to the loss of potassium in the sweat.

We have studied this problem fairly extensively and the sweat potassium concentrations are a little higher in cystic fibrosis of the pancreas than in the normal. In our series, I think the concentration in the patients was 10 mEq/L and the controls were 6 mEq/L, which means that one would really have to sweat an awful lot more than any of these children do to get potassium deficient in this way.

I think it is more likely that when they are ill, they have a fair amount of vomiting and may well lose potassium in this way, and then become somewhat alkalotic and lose potassium in the urine as part of the mass sodium effect.

DOCTOR ELIEL: I have one question which is addressed to the problem of the relationship between tissue and serum potassium levels and clinical manifestations.

Doctor Darrow has indicated that it will take several days to replete the potassium depleted tissue, and yet the clinical recovery from clinical manifestations of potassium deficiency occurs much more rapidly than that, in patients with diabetic acidosis and respiratory difficulties, for example.

What is the significance of the serum levels, the electrocardiograph changes, symptomatic manifestations and the actual tissue content of potassium? Doctor Relman, could you discuss this point for us?

DOCTOR RELMAN: That is a tough question. It has been our experience that in states of potassium deficiency with hypokalemia, the electrocardiogram is not a very accurate guide to the level of serum potassium and the degree of depletion. The serum level itself, on the other hand, is not a very accurate guide to the level of tissue depletion because many factors other than the total amount of potassium loss can change the serum level so that you cannot tell from the electrocardiogram, reliably, how depleted your patient is and you cannot tell from the serum level. Furthermore, the serum level and the electrocardiogram are only poorly correlated. This is in potassium-depleted states.

What about the other symptoms in potassium-depleted states; what explains the paralysis or the weakness; what exactly explains the renal lesion; is it the serum level or the tissue level? I don't think that one can draw any accurate relationship between any of these things.

Neuro-muscular function is affected by many different cations and many physical factors. We have to consider calcium, sodium, magnesium—many things that are not ordinarily measured simultaneously. We have to consider the rate of potassium depletion.

All these things play a role, and it is my experience that it is utterly impossible to know which patients are going to be paralyzed and which patients are not. I am constantly being surprised at the serum potassium levels. Patients will walk in. and clinically they don't look so bad—they have no reflex changes, their muscular strength is normal, and the serum potassium comes back 1.8 or 2.1 mEq/L.

Conversely, patients will come in with many of the symptoms which one would associate with potassium depletion, and the serum potassium may not be as low. So that I think one can make no hard and fast correlation.

On the side of hyperpotassemia, I think it is quite clearly shown by Doctor Tarail's studies and by the work of many people since, that the electrocardiogram in potassium intoxication responds to the serum level. It doesn't matter what the tissue level is—your patient will die of potassium intoxication when the serum potassium is 9 or 10 or 11 mEq/L., regardless of whether his tissues are overloaded with potassium or not. This is an acute thing which has to do with the extracellular concentration. I think one can forget about the tissues with acute potassium intoxication as far as the electrocardiogram is concerned.

When the electrocardiogram tells you that the patient has potassium intoxication, it is no sense to argue that the patient really has potassium depletion of his tissues and therefore he really needs more potassium. He will be just as dead of ventricular fibrillation, even though his tissues are depleted, as he would be if his tissues were normal.

DOCTOR ELIEL: Doctor Tarail, do you have any further comment about electrocardiograms and potassium?

DOCTOR TARAIL: I agree with Doctor Relman's splendid analysis of the various facets of the question, and I might just add a few things. Before you treat the patients with potassium, you must be sure that the patient's serum potassium is normal or low. Initially, in these patients, before rehydration, the serum potassium may be high in the face of a tremendous depletion of intracellular potassium; and, as Doctor Relman hinted, to fly in the face of a high serum potassium and administer potassium may result in a fatality.

In regard to paralysis, I think it is worth mentioning, to increase the puzzle, that it occurs also in patients with potassium intoxication.

Finally, electrocardiographic changes of potassium intoxication in man may occur at concentrations of serum potassium between 6 and 7 mEq/L.[4,5] This again emphasizes a more limited margin of safety than is implicit in a policy of cavalier administration of potassium to replenish supposed tissue deficit of the ion.

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Aldosterone and Potassium Metabolism

ROBERT E. COOKE

In 1873 von Bunge[1] first described the inverse relationship of change in balance of sodium and potassium. The ingestion of potassium citrate or bicarbonate increased the excretion of sodium. Likewise an increase in sodium intake led to an increase in potassium excretion. The validity of these early experiments has ben confirmed many times.[2,3] Winkler and Smith[4] demonstrated that an intravenous load of potassium chloride led to an almost immediate rise in sodium and bicarbonate excretion. Leaf and Couter[5] showed that the intravenous administration of sodium salts, particularly sodium bicarbonate, was followed by a marked rise in potassium as well as sodium excretion. Obviously such a phenomenon under certain dietary conditions could lead only to depletion of the other cation unless some homeostatic mechanism was present to prevent such an occurrence.

It is the purpose of this paper to review the renal mechanisms responsible for the "Bunge phenomenon" and the mechanisms compensating for these changes through the secretion of aldosterone. Neither new nor original concepts are presented.

Sodium Excretion

Although alterations in glomerular filtration rate may lead to changes in sodium excretion, variations in tubular reabsorption are primarily responsible for fluctuations in the amount of sodium in the urine.[6] Sodium reabsorption is currently considered to result from exchange of cation of intracellular origin for intraluminal sodium.[7] Hydrogen ion which comes mainly from the dissociation of hydrated carbon dioxide is probably the principal cation exchanged throughout the proximal tubule. The quantity of sodium reabsorbed per unit time would seem to be limited by the supply of hydrogen ions from the renal tubular cells. In the more distal region of the tubule intracellular potassium may also be exchanged for the sodium reaching this site.

Potassium Excretion

Although the preliminary studies of Bott[8] indicated that filtered potassium was almost completely reabsorbed in the proximal tubule, more recent micropuncture studies by the same worker[9] demonstrate that a small but significant quantity of filtered potassium escapes reabsorption in the proximal tubule. If non-selective reabsorption of sodium and potassium occurs in the proximal tubule, 5 to 15 per cent of filtered potassium may reach the distal tubule. In addition potassium is exchanged for filtered sodium in the distal tubule. The latter process probably accounts for the major portion of excreted potassium. Increase in potassium excretion may be observed both when sodium excretion increases and decreases. Such a paradox is best explained by assuming that two factors are operative in potassium exchange: (1) the number (and possibly the concentration) of sodium ions reaching the exchange site—"the mass factor;" (2) the degree of substitution of potassium for a given number of sodium ions—"the percentage factor." If sodium diuresis occurs, potassium output increases (unless potassium exchange is blocked as in mercurical diuresis), since statistically the opportunity for contact with exchange sites is greatly increased. If exchange sites are more accessible to sodium ions and the supply of intracellular potassium to these sites is abundant, exchange of potassium increases and effects a decrease in sodium excretion. Limitation of sodium intake markedly restricts potassium loss even when large doses of desoxycorticosterone are administered to rats.[10] Administration of desoxycorticosterone to rats on a fixed sodium intake increases potassium excretion and decreases sodium excretion.

Interrelationships of Sodium and Potassium Excretion

When the potassium concentration of the plasma reaching the renal tubule is increased intracellular pH probably rises,[11] thereby reducing the availability of hydrogen ions for exchange with sodium. Sodium reabsorption decreases and sodium excretion increases. The increase in potassium excretion under these circumstances has been interpreted as resulting from a shift in the equilibrium in which potassium and hydrogen compete for exchange at the same level of the tubule.[7] Telelogically it would seem more reasonable to assume that the greater mass of sodium reaching the distal tubule after potassium loading increases absolutely the exchange of cell potassium for intraluminal sodium. Since potassium excretion is almost totally dependent on exchange for sodium such a mechanism would guarantee an increased capacity for renal excretion of potassium when the plasma potassium level rose.

An administered load of sodium increases potassium excretion acutely in the same way. Whenever the accompanying anion is reabsorbed to only a limited extent (such as phosphate, sulfate or bicarbonate) the quantity of sodium reaching the distal tubule is increased and potassium excretion also rises. Diuretics such is Diamox® or Chlorothiazide® probably increase potassium excretion in this way. However, kaliuresis is limited after the administration of mercurical diuretics because exchange of potassium is blocked by these compounds.

Compensatory Mechanisms

It is obvious that some homeostatic mechanism is necessary to prevent depletion of body sodium when potassium is given or potassium when sodium is given. That aldosterone represents such a feedback system can be concluded from a review of the action of this hormone and of the control of its secretion.

Action of Aldosterone

The effects of aldosterone on various organs are listed in Table 1. The predominant action is on the kidney tubule. Injection of 40 micrograms intravenously into a patient with Addison's disease leads to a rapid (less than one hour) decrease in sodium excretion.[12] The excretion of potassium increases and therefore a marked fall in the sodium to potassium ratio of urine occurs.[13] Urinary titratable acidity and ammonia excretion also increase. The increase in ammonium ions in the urine probably results from trapping of ammonia by hydrogen ions as the

PROBABLE ACTION OF ALDOSTERONE

Kidney
Increase Exchange
H for Na
K for Na
Gastrointestinal
Increase Exchange
Stomach: H for Na
Colon: K for Na
Sweat and Salivary Gland
Decrease Na in precursor fluid
or
Increase Na reabsorption in duct
Muscle
Increase Exchange
H_i and K_i for Na_e
Leukocyte
Increase Exchange
K_i for Na_e

Table 1

exchange of hydrogen for sodium is increased. Ammonia production by the tubules is probably not increased since renal glutaminase activity remains normal after adrenalectomy even though ammonium excretion falls.[14] Exchange of sodium for hydrogen or potassium in other tissues is probably affected by aldosterone. Output of hydrogen ion by the stomach[15] and exchange of potassium for intraluminal sodium in the intestine[16] are increased by aldosterone. Furthermore aldosterone reduces the sodium concentration of sweat and saliva and decreases the sodium to potassium ratio in these secretions.[17] It is also likely that the distribution of sodium and potassium within the body may be altered since adrenal steroids including desoxycorticosterone increase the sodium content of leukocytes in vitro and decrease intracellular potassium concentration.[18]

The fundamental mechanism by which aldosterone affects transport or exchange is unknown. It is possible that hormones possess a somewhat similar mechanism of action, analogous to the action of all vitamins as coenzymes. Insulin is thought to affect membrane permeability to glucose;[19] pitressin membrane permeability to water.[20] The observed action of aldosterone could be explained by assuming that the hormone affected membrane permeability to sodium. In the kidney tubule aldosterone might thereby permit greater penetration if intraluminal sodium into cellular exchange sites for hydrogen and potassium.

The Control of Aldosterone Secretion

Aldosterone output is affected by a number of factors and at present no single regulating mechanism can be identified. The study of this problem is complicated by the fact that only a small fraction of the total amount secreted can be recovered in the urine. Studies in which aldosterone is injected indicate that only 0.1 per cent of the administered dose is recoverable in the urine when extracted at pH 3.[21] When pH is lowered to 1 hydrolysis of certain excretory products occurs and the total recovered rises to 3 to 5 per cent. The study of secretory rate is also complicated by the fact that variations in inactivation by the liver occur.[22] Measurement of aldosterone levels in the peripheral blood is not feasible at present. Catheterization of the adrenal vein permits a direct estimate of production. However, small errors in measurement of blood flow alter considerably the calculation of daily secretory rate.

Although aldosterone output continues after hypophysectomy, ACTH increases somewhat the secretion of this hormone. Perfusion of the isolated beef adrenal with fluid containing ACTH leads to a rise in salt retaining activity of the venous effluent.[23] Administration of ACTH to the intact dog also increases the aldosterone level of adrenal vein blood. In the human a significant and moderately sustained increase in alderstone output occurs after ACTH administration when sodium intake is limited.[12] When ACTH is administered to a patient on a high sodium intake, there is a brief increase in urinary aldosterone, a decrease in urinary sodium, an increase in potassium excretion and an increase in body weight followed by a return of urinary aldosterone to control levels.

On the basis of transection experiments a neurohumoral mediator from the hypothalamus has been postulated.[24] However, these experiments are difficult to interpret

because of complicating factors such as blood loss, shock, anoxia, transfusions, etc., that may affect the adrenal directly.

Elevation of the serum potassium concentration leads to an increase in the aldosterone secretion. Perfusion of the isolated beef adrenal with a solution containing 25 mEq. per liter of potassium led to a significant increase in aldosterone concentration in the adrenal venous effluent.[23] In the intact animal that is sodium depleted potassium loading leads to a rapid rise in serum potassium concentration which in turn evokes an increase in aldosterone secretion.[25] When the intake of sodium is adequate, potassium loading produces neither a rise in potassium concentration nor an increase in aldosterone. In a patient with heart failure, edema and hyperpotassemia reduction of the serum potassium by means of Mercuhydrin® and a low potassium diet led to a fall in aldosterone excretion from 300 micrograms per day to 35. Water loading during pitressin administration increases total body water and lowers plasma sodium and potassium concentrations. Increase in dietary potassium chloride raises plasma potassium and aldosterone output.

The volume of body fluids may have even greater effect on aldosterone secretion than the concentration of potassium in plasma. The restriction of sodium intake with loss in body weight and body water leads to an increase in aldosterone secretion. Restoration of sodium to sodium-depleted subjects is followed by increases in body weight and body water and decreases in aldosterone output. This effect is not dependent upon changes in total body water but rather to changes in the extracellular space. Administration of hypertonic saline decreases aldosterone output even though intracellular water and total body water decrease. The blood volume affects aldosterone output rather than interstitial volume. Removal of blood with replacement by isotonic saline causes an increase in aldosterone secretion even though body weight and interstitial volume increase.[12] Removal of blood with reinfusion of cells in salt poor albumin causes a decrease in aldosterone output even though body sodium is diminished.

The Balance Between Immediate and Delayed Response to Electrolyte Loads

Such concepts of aldosterone action and control provide an explanation for the maintenance of relatively constant body composition despite wide fluctuation in electrolyte intake. When a load of sodium is administered, potassium excretion increases immediately. The excreted potassium comes from renal tubular cells[26] which in turn remove potassium from the plasma. The fall in plasma potassium concentration as well as the rise in effective circulating volume caused by the intake of sodium decreases adrenal cortical secretion of aldosterone. If renal tubular permeability to sodium is decreased by a fall in aldosterone levels, exchange of hydrogen and potassium for sodium decreases and sodium excretion rises as potassium excretion falls. Likewise reduced exchange of plasma potassium for sodium in the gastrointestinal tract tends to restore body composition toward normal.

On the other hand depletion of sodium by extrarenal routes such as in diarrhea leads to marked reduction in the quantity of sodium reaching exchange sites for potassium in the distal tubules particularly if glomerular filtration rate is lowered by concomitant circulatory insufficiency. The fall in extracellular volume and probably rise in potassium concentration under these circumstances effects an increase in aldosterone secretion. The elevated levels of aldosterone not only permit conservation of sodium but also facilitate potassium excretion. Increased gastrointestinal conservation of sodium and excretion of potassium also occur.

When a load of potassium is administered, sodium loss occurs immediately and depletion of sodium would result except for a rise in aldosterone output. The increase in plasma potassium concentration that results from such a load as well as the reduction in extracellular volume from sodium loss increases aldosterone secretion. Elevated levels of aldosterone increase sodium conservation and facilitate the excretion of potassium by increasing exchange in the distal tubule. The action of aldosterone on the gastrointestinal tract limits potassium absorption and conserves body sodium.

Depletion of potassium likewise reduces aldosterone output, potassium exchange in the renal tubule and potassium loss into the gastrointestinal tract thereby tending to restore body composition toward normal.

Conclusion

The immediate effect of loads of sodium or potassium is an increase in the urinary excretion of the other ion. Depletion of the ion the supply of which is limited is prevented by alterations in the output of aldosterone.

The mechanisms controlling the excretion of sodium and potassium and the secretion of aldosterone are discussed.

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DISCUSSION

DOCTOR ELIEL: Why do patients with primary aldosteronism fail to exhibit edema?

DOCTOR COOKE: I have no notion why. I would think that usually primary aldosteronism is associated with hypertension. I wonder whether or not there could be increased filtration rate and some loss of sodium in this way. I think this might be a possible explanation.

DOCTOR ELIEL: Doctor Relman, do you have anything to add?

DOCTOR RELMAN: I don't know the answer either, but I think the analogy between primary aldosteronism and normal patients given prolonged high doses of salt-retaining steroids is pretty good.

Now, if you take a normal man and give him Desoxycorticosterone or some other sodium-retaining steroid in high doses for a long period of time, you cannot produce edema. You produce first a phase of sodium retention which rapidly passes despite continued administration of the steroid, and the subject rapidly comes back into normal sodium balance.

This, I think, is the situation that we see in patients with primary aldosteronism. At the same time, there is a tendency for the glomerular filtration rate to rise in normal subjects given steroids, and this may be part of the mechanism which enables them to escape from the sodium-retaining action of the steroid. That there is such a mechanism, I think, is quite clear.

If you consider secondary aldosteronism, for example, someone who has edema from low plasma protein, from say the nephrotic syndrome or someone who has edema from congestive heart failure and an increased aldosterone output, you have a situation in which retention of sodium does not enable the renal circulation or the glomerular filtration rate to get the full effect of that retention because of the fact that

the sodium is being sequestered in the tissues. As far as the essential circulation is concerned, that sodium is not being retained—it is being lost into the tissues as a result of the high capillary pressure or the low colloid osmotic pressure. The idea that I am suggesting is simply that in primary aldosteronism, compensatory mechanisms, which may include increased glomerular filtration rate, come into play because the sodium remains in the circulation. In secondary aldosteronism, compensatory mechanisms do not come into play because that sodium is leaking out of the circulation into the tissues.

DOCTOR COOKE: Actually, I think one does see edema from salt-retaining hormones, but you have to look at a different species from the one Doctor Relman works in—namely, small babies. You will see edema develop under those circumstances very readily and each stress by itself actually will precipitate edema during their salt-retaining phase.

DOCTOR TARAIL: Perhaps I will have the courage to suggest an alternative possible explanation, although I suppose I will be stoned or throttled by my colleagues here. Perhaps this fact that patients with primary aldosteronism do not develop edema in the face of a tendency toward sodium retention is evidence against the idea that sodium has a great deal to do with it. Perhaps this is evidence against the idea that sodium retention is importantly involved in edema.

Acute Renal Insufficiency

BEN I. HELLER

NUMEROUS clinical disorders associated with transient hypovolemic shock, dehydration, depletion of sodium and chloride, metabolic acidosis, and trauma may precipitate varying degrees of decreased renal function and result in oliguria. If these disorders are promptly recognized and correctly treated, renal blood flow may be quickly restored to normal and anatomic alterations of the renal tubules will not occur. On the other hand, these same disorders of a more severe and persistent degree may cause an intense and persistent decrease in renal blood flow and acute renal insufficiency may ensue. During the past fifteen years a syndrome of acute renal insufficiency associated with a wide variety of precipitating factors has become recognized with increasing frequency. It is of the utmost importance that the syndrome be recognized early and proper treatment instituted promptly since errors in diagnosis and treatment may lead to the death of the patient.

Acute renal insufficiency may be defined as a syndrome of rapid but *reversible* loss of renal function due to renal ischemia or nephrotoxic agents and resulting in necrosis of cells of the proximal tubules and disseminated areas or tubular necrosis with disruption of the basement membrane in both the proximal and distal nephron.[1] Synomyms that have been used are acute renal failure, acute tubular necrosis, shock kidney, crush syndrome, hepatorenal syndrome, toxic nephrosis, hemoglobinuric nephrosis, burn nephritis, acute parenchymatous degeneration, extra-renal uremia, and lower nephron nephrosis. The term "lower nephron nephrosis," which implies localization of pathologic change to the distal nephron, is a poor one despite its wide acceptance, since the renal lesion is disseminated. The terms "acute renal insufficiency," "acute tubular necrosis," and acute renal failure appear to be gaining wider acceptance in current literature.

Pathologic Physiology

The majority of patients with acute renal insufficiency will have experienced shock due to blood loss, dehydration, or bacteremia. In those patients who do not manifest the clinical signs of peripheral vascular collapse, hemoconcentration and a deficit in circulating blood volume will frequently be present. The studies of Lawson and associates[2] indicate that the kidney participates to a pronounced degree in the vasoconstrictive phenomena of shock. The decrease in renal blood flow is considerably greater than the reduction in cardiac output, and the glomerular filtration rate and tubular function are greatly reduced.[3] Anatomic alterations of the renal tubules consisting of disseminated areas of tubular necrosis and disruption of the tubular basement membrane result from the renal ischemia and hypoxia.

Hemolytic transfusion reactions and other disorders associated with a hemolytic process are commonly followed by acute

renal insufficiency. The distal tubular lumina and collecting ducts are often obstructed by hemoglobin casts, but obstruction per se is not an adequate explanation for the oliguria. In some instances hemolytic transfusion reactions are associated with peripheral vascular collapse. Even when this is not present, however, the precipitation of the casts appear to be secondary to intense renal vasoconstriction, marked reduction of the glomerular filtration rate, and oliguria. In the crush syndrome, in which large amounts of myohemoglobin are released from striated muscle, renal insufficiency is produced through a similar mechanism.

Renal tubular necrosis is caused by numerous substances. Chief among these are carbon tetrachloride, mercuric bichloride, and the sulfonamides. The reduction in renal blood flow has been demonstrated to be secondary to the tubular damage produced by the nephrotoxic agent. The pathologic lesion produced by such agents is chiefly a necrosis of the proximal nephron cells without disruption of the basement membrane.[1]

The marked reduction in glomerular filtration rate associated with the intense renal ischemia is undoubtedly a factor in producing oliguria. Back-diffusion into the renal interstitial tissue of that glomerular filtrate which is being formed is probably the most important factor. Experimental evidence for the unselective back-diffusion of the glomerular filtrate across the damaged tubular membrane was first noted by Richards.[4] When phenol red was injected into the ureter of a frog's kidney immersed in oxygenated Ringer's solution, the phenol red remained concentrated in the loop of Henle. When hydrocyanic acid was injected into the kidney, the dye no longer remained concentrated in the tubules but diffused out into the surrounding renal tissue. Richards also noted that selective tubular function was abolished in the frog kidney which was poisoned by mercuric bichloride. He found that the glomerular filtrate appeared to be increased but no urine was formed. He concluded that the damaged tubular epithelium allowed the filtrate to be drawn back into the blood stream by osmotic pressure. The demonstration of disruption of the tubular cell basement membrane, the decreased $\frac{U}{P}$ ratios for creatinine and inulin and negative $T_{m\ pah}$ values are all in keeping with this concept.

Clinical Course

The clinical course of acute renal insufficiency is extremely variable—the degree of variability depending upon the nature of the initial illness or injury, the degree of renal insufficiency and the rate with which it develops, the type of therapy, and the rate of recovery in renal function. In spite of the great variability in the clinical course, the syndrome may be divided into the following three phases: 1) shock, 2) acute renal insufficiency or period of oliguria, 3) the period of recovery in the early and late phases of diuresis.

The duration of the first phase will depend upon the promptness and adequacy of the treatment of shock. If dehydration is present, as it may be in patients during surgical operations, the volume of blood loss which may precipitate peripheral vascular collapse may be less than that causing shock in normally hydrated patients. Other symptoms and signs will, of course, depend upon the primary disease and associated conditions. Examples of such disorders are traumatic or operative shock from blood loss, post-partum or post-abortional bleeding, diabetic acidosis, and severe diarrhea. It must be empasized that shock may be absent according to the usual clinical criteria, but water and electrolyte losses may result in appreciable decreases in plasma volume and marked reduction in renal blood flow.

Following shock, hemolytic transfusion reactions, or the ingestion or inhalation of nephrotoxic substances there is a rapid development of progressive renal insufficiency. This is frequently ushered in by fever, nausea and vomiting, abdominal distention, occasionally jaundice, and pain in the costovertebral angle. Diminution in urinary output to 50-400 ml. per day is the general rule. The syndrome may develop in

spite of a daily urine volume of 400-1000 ml. per day. This was found in two of the 25 consecutive cases in our series (Table I).

DURATION OF OLIGURIA—25 CASES

Diagnosis	No. of cases	Days of oliguria	Deaths
CCl_4 Poisoning	12	3-15	2
Hypovolemic shock	6	6-15	4
Hemolytic transfusion reaction	3	1-7	1
Acute pancreatitis	2	6-11	1
Staphylococcal bacteremia	1	0	
Unknown (severe alcoholism)	1	9	1

TABLE I

The urine may be grossly bloody or smoky for the first few days; it is acid in reaction and contains large amounts of protein. The initial urine may have a high specific gravity but concentrating ability is rapidly lost, and the specific gravity becomes fixed at 1.010 to 1.012. The urinary sediment initially contains red blood cells, white blood cells, pigment casts, and hyaline and granular casts. The erythrocytes soon disappear from the urine, the proteinuria diminishes, and varying numbers of leukocytes may be seen.

As the oliguria persists, there is a marked tendency for the development of signs of congestive heart failure with pulmonary edema. This is especially true if salt and water are administered injudiciously. Cardiac dilatation, cardiac overactivity with loud heart sounds, gallop rhythm, elevated venous pressure, and normal or rapid circulation times are noted. The cardiac failure is of the high output type.[5] It is questionable whether a metabolic defect in uremia is responsible for the heart failure. In some instances the signs of congestive heart failure will resolve in the early phase of diuresis although there is a progression in the uremic syndrome. It is customary to administer digitalis to these patients, and we have followed this custom; but the efficacy of digitalis is questionable. There have been no definitive studies relative to the effects of digitalis either in this syndrome or in the high output failure of acute glomerulonephritis. When congestive heart failure develops, an analysis of fluid therapy and daily weights will usually reveal unmistakable evidence of overhydration. In an occasional patient, however, pulmonary edema will develop in spite of judicious therapy. It is conceivable that increased capillary permeability may play a role in this phenomenon.

Hypertension usually develops in the first few days, the systolic hypertension being more pronounced than the diastolic in most cases. In two of our patients there was a wide fluctuation in blood pressure from slight elevation to levels as high as 256 mm. mercury systolic and 140 diastolic. Eight of our 25 patients had normal blood pressure.

Gastrointestinal symptoms may continue in the oliguric period with abdominal distention, nausea and vomiting, and diarrhea. These symptoms, as is true of all others, may vary greatly from patient to patient. Two of the patients in this series had massive gastrointestinal bleeding. One patient had carbon tetrachloride poisoning in which the bleeding diathesis is generally more pronounced. One patient with hypovolemic shock sustained an episode of massive hematemesis on the sixth day of diuresis. Although this was promptly treated with blood transfusion and the period of shock was transient, oliguria recurred and death followed five days later from uremia and hyperpotassemia.

Cerebral symptoms are common and are similar to those seen in any patients with uremia. Restlessness, apprehension, delerium, and coma may be present. We have been impressed with the extreme apprehension, restlessness, and the expression of fear in patients with hyperkalemia. When convulsions occur, they will usually have been preceded by the administration of excessive amounts of salt and water.

Early deaths are usually due to the primary disease, but fatal pulmonary edema may occur early if incorrect therapy is given. Deaths later in the oliguric phase are

due principally to pulmonary edema, uremia, and hyperpotassemia (Table II).

CAUSES OF DEATH

Days of Oliguria	Day of Death	Cause
11	11	Pulmonary edema
8	8	Pulmonary edema
9	9	Pulmonary edema
3	7	Uremia
6	15	Uremia
15	15	Uremia, pulmonary edema
10	10	Hyperpotassemia
7	10	Hyperpotassemia
8	8	Uremia

TABLE II

Laboratory studies reveal a rapidly developing normocytic, normochronic anemia. The hemoglobin falls to 8 to 11 gms. per 100 ml. of blood within the first 10 to 14 days after the inception of oliguria. It then stabilizes at this level even though the oliguria may continue. The anemia is due to both decreased erythropoiesis and hemolysis. The anemia may persist for many weeks or months after recovery. In one patient the hemoglobin was 8.4 gms. per 100 ml. of blood during the phase of acute renal insufficiency. Five months later, at a time when renal function had returned to normal, the hemoglobin was 9.9 grams, and the anemia was unresponsive to iron. Since the anemia is well tolerated and since blood transfusion in the oliguric period may precipitate pulmonary edema, it is believed that blood transfusion is contraindicated.

Rapidly rising blood urea, decreased serum sodium, chloride, calcium and bicarbonate; and varying elevations of serum inorganic phosphorus and potassium are noted. Lowering of the serum sodium can frequently be accounted for by the dilution of body fluids by the parenteral administration of sodium-free fluids. In some patients, however, hyponatremia appears to be caused by intracellular shifts of sodium or extracellular shifts of water.

Potassium commonly rises to levels that are neurotoxic and cardiotoxic. The effets of hyperpotassemia are accentuated by coexistent hyponatremia, hypocalcemia, and metabolic acidosis, paresthesias of the extremities, muscular weakness, pain, and tenderness; flaccid peripheral paralysis and respiratory paralysis may occur when the serum potassium is above 8 mEq. per liter We have been impressed with the marked degree of apprehension, restlessness, and cloudiness of the sensorium which occur. Serial determinations of serum sodium and potassium and serial electrocardiogram are essential in following progress. The first electrocardiographic abnormality is the presence of high, peaked T waves with a narrow base, noted initially in the precordial leads and then in the limb leads. These are usually seen first in precordial lead V_2. This finding may quickly be followed by depression of the ST segment, varying degrees of auriculoventricular and intraventricular block, disappearance of the P wave, bradycardia, periods of asystole, and varying types of ventricular arrhythmias. It has recently been shown that elevation of the serum magnesium, even when the serum potassium is only slightly elevated, may give rise to the same signs and symptoms.[6]

Diuretic Phase

The average duration of the period of oliguria is 10 to 12 days but it may persist for as long as 21 days. The duration of oliguria in our patients varied from 3 to 15 days. As tubular regeneration occurs, there is a gradual increase in urine volume. Rapid and marked weight loss is noted with the onset of diuresis even in those patients who are not clinically overhydrated during the oliguric phase. The data of Swann and Merrill[7] indicate that the diuresis is related in part to retention of water during the phase of oliguria. This represents water of oxidation and the release of bound tissue water. The oxidation of one gram of carbohydrate, protein and fat will yield 0.55 ml., 0.41 ml., and 1.07 ml. of water respectively. The average daily fat consumption for two patients reported by Swann and Merrill was 310 grams and 255 grams. The average daily protein consumption was 48 grams and 22 grams. It is the contention of these authors that the quantitative replacement of fluid

and electrolyte losses during the diuretic phase results in a continuous, forced diuresis. There are, however, some patients in whom diuresis will be so intense that depletion of extracellular fluid and diminished blood volume will lead to circulatory collapse is adequate fluid and electrolyte replacement is not instituted. This occurred in two patients in this series.

The blood urea may continue to rise for several days after diuresis occurs. This is usually not cause for alarm, and steady improvement in the clinical course follows correct therapy. In other instances, however, symptoms and signs of acute renal failure will progress in spite of diuresis. The uremic syndrome may become intensified, and hyperpotassemia may progress. Twenty to 25% of the deaths reported in the literature occur during the diuretic phase. Of the 9 deaths among our 25 patients, three occurred during this phase.

Data relative to the urinary concentration of sodium and potassium is available for 10 patients. The mean urinary sodium concentration was 38 mEq. per liter with a range of 17-72 mEq/liter. The mean urinary potassium concentration was 27 mEq. per liter with a range of 20-31 mEq. per liter.

After clinical recovery normal renal function may not be reestablished for several months.[8] The ability to excrete a concentrated urine is the last function to return to normal.

Differential Diagnosis

The following diseases must be considered in differential diagnosis: Acute glomerulonephritis, acute exacerbation of chronic glomerulonephritis, acute pyelonephritis, ureteral obstruction from stone, sulfonamide crystalluria, metastatic cancer with ureteral obstruction, and renal vascular lesions such as malignant hypertension and renal infarction. The differential diagnosis of these diseases is usually not a difficult problem. As a general rule, in the absence of a history of a disease or an event known to precipitate acute renal insufficiency, the diagnosis will usually prove to be another form of renal disease. Anuria is rare in acute renal insufficiency. It is more commonly found in acute glomerulonephritis and in complete obstruction of the ureters.

Given a disease or a condition known to precipitate acute renal insufficiency, the urine volume should be carefully evaluated following blood replacement or electrolyte and water repletion. If oliguria persists following these therapeutic measures, a diagnosis of acute renal insufficiency may then be made.

Treatment During the Phase of Oliguria

The keynote of therapy during the oliguric phase of acute renal insufficiency is the rigid restriction of fluid intake. It has been estimated that the average insensible water loss from the skin and lungs of an afebrile adult patient is approximately one liter. Endogenous water formation from tissue catabolism and the release of bound water has been estimated to be approximately 450 millileters per day. Since these figures are only approximations, the only reliable guide to proper fluid therapy is the daily body weight. In view of the fact that basic caloric requirements can usually not be provided to the oliguric patient, proper fluid balance should be accompanied by a small daily weight loss. This usually amounts to between one-tenth and four-tenths of a kilogram daily. Weight loss greater than .5 kilograms probably indicates dehydration. Maintenance of body weight or an actual gain in weight indicates overhydration. In our experience the usual daily fluid requirement has varied from 400 to 550 milliliters per day. This has been administered in the form of 50% glucose given via a polyvinyl catheter which has been threaded into the basilic or femoral vein. The degree of inhibition of tissue catabolism from the parenteral administration of glucose is variable. When acute renal insufficiency follows severe trauma or postoperative shock, the degree of inhibition does not appear to be very marked. On the other hand we have noted in patients with carbon tetrachloride poisoning that considerable protein sparing is achieved by this amount of parenteral glucose.

Oral alimentation should never be attempted early during the oliguric phace. Indeed, in our own experience alimentation attempted by oral routes even during the latter phase of oliguria frequently meets with failure. Various types of fat emulsions have been used with varying degrees of success. When fat emulsions are given orally even during the latter phase of oliguria nausea and vomiting and diarrhea are common occurrences. In only one patient in this series was a fat emulsion used successfully throughout the phase of oliguria. The administration of protein or potassium in any form is, of course, absolutely contraindicated during the oliguric phase.

There exists a considerable difference of opinion about the treatment of hyponatremia. Hyponatremia commonly follows the excessive administration of sodium free fluids. This is more suitably treated by the restriction of water rather than by the administration of sodium. When hyponatremia develops as a result of loss of sodium by vomiting, diarrhea, or excessive sweating, there can be no question that such sodium deficits should be replaced. When sodium deficit and metabolic acidosis coexist, it is advisable to replace the calculated sodium deficits with both hypertonic sodium chloride and sodium bicarbonate. Because of the ever present danger of precipitating pulmonary edema, such sodium deficits should be replaced cautiously. In some instances hyponatremia will develop although there is no evidence of exogenous loss or overhydration. In such instances, and when serious hyperkalemia (serum potassium above 7 mEq. per liter) is not present, we have not attempted to correct the hyponatremia. Moreover, diuresis is not inhibited by low levels of serum sodium. Diuresis was noted to occur in several patients when the serum sodium was below 126 mEq. per liter. In one patient the diuretic phase began when the serum sodium was 117 mEq. per liter.

Therapeutic measures to lower the extracellular level of potassium are indicated when the serum concentration of this ion has reached levels of 6.0 mEq. per liter. In some instances the simple addition of regular insulin to the 50% glucose (one unit per three grams of glucose) will be sufficient to lower the serum potassium and restore electrocardiographic abnormalities to normal (Figure 1). Such therapy may occasionally suf-

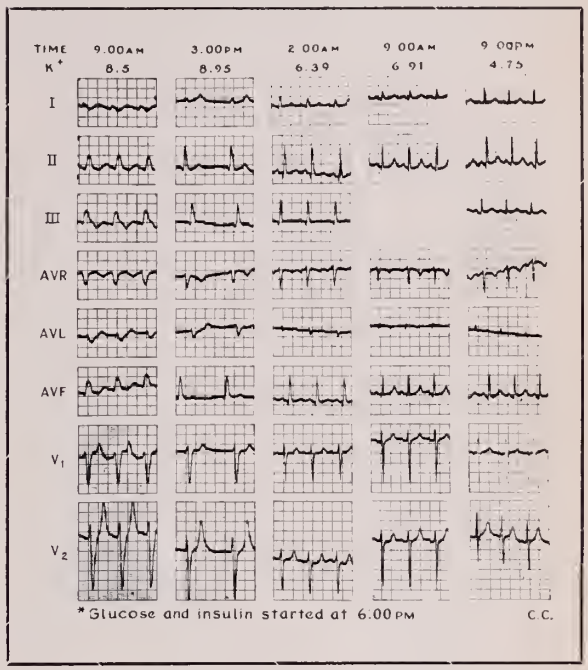


Figure 1. Effect of glucose and insulin on hyperkalemia and electrocardiographic abnormalities.

fice until the diuretic phase begins. On the other hand, if catabolic processes are marked, lowering of the serum potassium may be only transient. If the effects are transient and metabolic acidosis, hyponatremia, and hypocalcemia are contributing to the effects of the hyperpotassemia, calcium gluconate and sodium lactate may be given in the constant intravenous infusion as part of the daily fluid requirements. (Calcium gluconate and sodium bicarbonate cannot be added to the same infusion since calcium carbonate will be precipitated.) The correction of hypocalcemia may cause the electrocardiographic abnormalities to revert to normal even without changes in the concentration of the elevated serum potassium.[9] Potassium-free ion-exchange resins may also be given in a 10 per cent suspension as a retention enema. In the majority of cases, hyperkalemia can be controlled by judicious therapy during the course of oliguria. In those instances in which this is not possible, hemodialysis is the treatment of choice.

Treatment During the Diuretic Phase

The onset of the diuretic phase has been arbitrarily defined as that period during which urine volume exceeds 400 ml. per 24 hours. The urine volume generally increases by step-wise increments, but in an occasional patient there will be noted a sudden increase to two or more liters per day.

The guiding principle of therapy in the diuretic phase is the replacement of the excessive renal water and electrolyte loss Guides to the adequacy of such replacement are the body weight, the serum and urinary concentration of electrolytes, the urine volume, and the clinical signs of depletion of the plasma and extracellular fluid volumes. In the latter regard, the development of orthostatic hypotension and tachycardia in the sitting position may be a useful sign to indicate hypovolemia due to salt and water deficit. Hypokalemia may occasionally develop and should be corrected by the addition of potassium to the parenteral fluids. When moderate or severe metabolic acidosis is present, sodium replacement by both sodium bicarbonate and sodium chloride will facilitate correction of the acidosis. Parenteral fluids should be given slowly since too rapid infusion, especially in the early phase of diuresis, may precipitate convulsions or pulmonary edema.

Restriction of water may decrease the degree of diuresis but not abolish it. In one patient with carbon tetrachloride poisoning the fluid intake was intentionally limited to 2500 ml. per day for two days. The urine volume, however, was approximately four liters per day for those two days. Negative water balance was associated with increasing azotemia. In two other patients inadequate water and electrolyte replacement was associated with hypotension and tachycardia. It is apparent that, in these patients, the diuresis was due to the defective tubular reabsorption of water and not to the excessive production of metabolic water or overhydration during the phase of oliguria. If edema is present or clinical signs of overhydration are present it is obvious that the fluid prescription should permit the removal of this excess fluid.

When the early diuretic phase is well established and there are no gastrointestinal symptoms, a low protein, high carbohydrate diet may be instituted. This is generally well tolerated even if azotemia is still present. Later in the diuretic phase the protein content of the diet may be increased.

Prognosis

When acute renal insufficiency is uncomplicated by coexistent disease or injury, the prognosis is improved. Thus, in the present series two deaths occurred in 12 patients with carbon tetrachloride poisoning whereas 7 deaths occurred in the other groups of complicated acute renal insufficiency. Patients with acute renal failure secondary to chemical toxins appear to have a milder clinical course and to regain tubular discriminatory function more rapidly than do those patients with complicating disease or injury. Since recovery in the latter group is conditioned by the severity of the associated disease or injury, mortality statistics in various series are not comparable.

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DISCUSSION

DOCTOR CONRAD: Doctor Heller, in view of the acidosis in acute renal failure, why wouldn't hypertonic sodium lactate be preferable to hypertonic saline?

DOCTOR HELLER: If there is an exogenous sodium loss associated with fairly severe metabolic acidosis, I think indeed it would be better to give either sodium lactate or sodium bicarbonate along with the sodium chloride than the hypertonic sodium chloride alone. This poses another problem, and that is when

should be metabolic acidosis be treated? I don't know of any good answer to that other than to follow the pH if such is available, or to treat when the patient becomes symptomatic from severe metabolic acidosis. In such an instance, of course, life can be threatened by the acidosis per se, and so we have played it purely by ear and have treated the metabolic acidosis by bicarbonate and saline when it has become essential.

DOCTOR CONRAD: Is the use of an oral or an intravenous fat emulsion of value in meeting caloric requirements and reducing protein breakdown in acute renal failure?

DOCTOR HELLER: Yes, I think it is. The difficulty of giving anything orally early is that most of these patients have nausea and vomiting. However, one of our patients with carbon tetrachloride poisoning was treated with the Borscht regime and had very little in the way of metabolic acidosis and a very smooth convalescence. In those patients who can tolerate oral fat, there is an indication that cellular breakdown can be diminished. The other part of the question dealt with intravenous fat. I have had no experience with intravenous fat at all.

DOCTOR RELMAN: I have had no experience, but a rapid calculation I made yesterday from the information Doctor Mueller gave us would suggest that if you want to get the most possible calories for the least possible volume, 50% glucose solution would be better than the solution he described to us yesterday. There would be slightly more calories per cc in 50% glucose.

DOCTOR CONRAD: Doctor Heller, there is a series of three questions here which relate to the treatment of acute renal insufficiency. The first is what is your opinion of intermittent peritoneal lavage as a means of therapy in acute renal failure? The second asks about hemodialysis in such a situation; and the third concerns the use of venosection and blood transfusions in acute renal failure with high blood urea nitrogen when dialysis is not available.

DOCTOR HELLER: I have had no personal experience with intermittent peritoneal lavage, but I think since dialysis has become available, this has been pretty well given up. The complications of peritoneal lavage are considerable. Infections were invariable and to those who have used it, posed considerable problems.

As to hemodialysis, I think there are real indications for this. I think the artificial kidney has proved its worth. One cannot compare mortality statistics in the various reports in the literature. For example, the mortality in the 85 cases reported by Swann and Merrill was over 46%. Not all of those cases were treated by hemodialysis. It is almost impossible to control the various variables in the individual cases. The patients may die from their primary disease even without the advent of acute renal insufficiency. Certainly hemodialysis can be life-saving in the event of hyperkalemia that is not responding to any other method of therapy.

If the questioner is suggesting venesection as a means of exchange transfusions, I think it has been fairly well demonstrated that this does not play any real part in the treatment of renal insufficiency.

As to transfusion of these patients, I think it has been demonstrated that they stabilize pretty well a level of hemoglobin of about 8 gm/100 ml. of blood. Transfusions carries with it increased plasma volume, with a threat of pulmonary edema, and moreover it carries with it increase in the serum potassium. I would not advocate transfusion of these patients who appear to tolerate their anemia fairly well.

I might comment for just one moment as to the so-called congestive heart failure. These patients, with the development of so-called congestive heart failure, have an increased venous pressure with a normal circulation time. This indicates that they have a high cardiac output. So far as I know, there are no physiologic data available on the function of digitalis in the congestive failure of patients with acute renal insufficiency. When digitalis is used, one should protect against the development of digitalis toxicity when these patients start to diurese or when they are subjected to dialysis.

DOCTOR CONRAD: Is it advisable to use insulin in connection with glucose in the treatment of acute renal failure?

DOCTOR HELLER: We have started using insulin with glucose when the serum potassium reaches 6.5 mEq/L. Whether it would make any difference if one used it throughout the course of renal failure, I don't know. We do use it routinely when the serum potassium reaches 6.5.

DOCTOR CONRAD: A member of the audience has made an observation concerning severe and resistant bacteremias in patients with acute renal insufficiency or acute tubular necrosis. He says in his experience this is one of the most serious problems in his patients and he wishes you to comment on whether or not you have observed such patients, and if so, what you do about it.

DOCTOR HELLER: Only one of the patients of the 25 that I presented had a staphylococcal bacteremia terminally. This was a postoperative patient and it originated in an intra-abdominal abscess. This has not been a problem in our other patients. It is true, however, that patients who develop this postoperatively do have poor wound healing and may develop infection on that basis. One of our patients, for example, had a fairly severe dehiscence of a wound; but other than that, we have not been troubled with various types of bacteremia.

The Recognition and Management of Fluid and Electrolyte Alterations in Surgical Patients

WILLIAM E. ABBOTT
HARVEY KRIEGER
STANLEY LEVEY

WITH THE IMPROVEMENT of techniques and the advent of antibiotics the mortality and morbidity accompanying surgical procedures have steadily decreased. However, the management of fluid and electrolyte problems in both medical and surgical patients still presents some difficulties. The confusions which exists in the minds of many physicians in regard to the proper way of managing fluid and electrolyte problems is based on the misconception that this type of therapy is complex, difficult, and is primarily dependent on the chemical findings in the blood and serum. Also some rather complicated laboratory procedures which have been employed to investigate these problems have been suggested for the clinical management of patients with fluid and electrolyte abnormalities. The following discussion is based on practical methods which can be used for recognizing and treating surgical patients primarily by bedside observations and examinations rather than by depending on the chemical laboratory or by doing complex metabolic balance studies.

Periodically over the past thirty years the blood count, hematocrit, whole blood specific gravity and plasma protein or electrolyte concentrations have been advocated as guides for correcting fluid and electrolyte imbalances. More recently the methods of determining the plasma or blood volume have been improved and simplified so that these measurements have also been advocated as a means of recognizing alterations in fluid balance. While all of these methods may be helpful in managing the patient, the data they provide may at times be confusing or fail to reveal fluid and electrolyte abnormalities.

The errors in the blood and serum chemical determinations are usually not due to improper analytical techniques but more frequently occur because of improper obtaining of specimens or not separating the cells from the serum soon enough. It should also be remembered that blood or serum concentrations do not give information as to the state of fluid, electrolyte or nutritional balance. The state of balance of the patient is only determined by measuring the intake of fluid and nutrients and checking this against the output of the same substances. Dehydrated untreated patients may show a high or even normal hematocrit, protein, sodium, potassium and chloride concentration. This occurs because the kidneys often maintain approximately normal blood concentrations in spite of the fact that dehydration and deficits of these substances may exist. Frequently patients with a low intestinal obstruction, an ileostomy or internal shifts of fluids and electrolytes may be markedly dehydrated without a significant change in their blood or serum chemistries.

When there is an increase in capillary permeability substances used for measuring plasma volume (dye or isotope) "leak" from the vascular compartments in abnormally large amounts and a false high value for plasma volume may be obtained when actually hypovolemia exists. Plasma volume studies are most valuable to denote changes when serial determinations are carried out. This is usually not feasible in therapeutic endeavors. Laboratory determinations can be of great assistance if the state of balance is estimated from the patients history and physical findings and assessed in light of the patients clinical course.

Management of fluid and electrolyte balance usually is quite simple when the composition and the magnitude of the internal shifts, external losses and the approximate daily requirements are carefully estimated. In the following discussion the ways in which these losses can be determined will be presented.

Normal Adult Intakes and Outputs—The water intake of a normal adult is about 2000 to 3000 ml. a day of which approximately one-half is in the form of liquids and the remainder is derived from the water which is contained in soft or solid food (800-2000 ml. per day) and the water of oxidation (200-500 ml. per day). The fact that solid foods are 60 to 95 per cent water is often over-looked and thus the intake of water in the form of solid foods is frequently not considered. While the intake of electrolytes may vary quite widely the average adult consumes about 80 to 160 mEq. each of sodium and potassium and from 150 to 280 mEq. of chloride. This intake of chloride, greater than either of the two cations, should be remembered in the management of patients with hyperchloremic acidosis following uretero-sigmoidostomies or renal disease.

Usually in a healthy individual ⅓ to ½ of the fluid and approximately 90 per cent of the electrolyte and nitrogen intakes are excreted by the kidneys each day. Normally an individual loses about 500 ml. of electrolyte-free water from the lungs and a comparable amount of water containing approximately 40, 5 and 25 mEq. per liter respectively of sodium, potassium and chloride in perspiration. These physiological losses and the concentrations of sodium, potassium and chloride in such fluids are summarized in Table 1 and shown schematically in Figure 1.

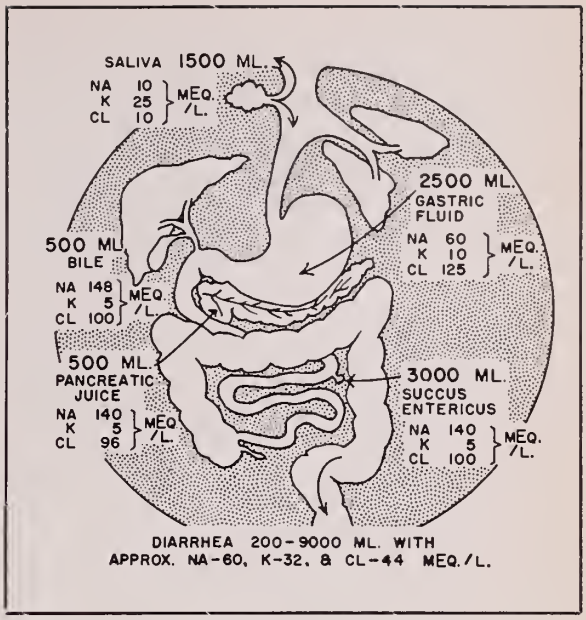


Figure 1. Daily physiologic losses of water and electrolytes.

Since fluid and electrolyte balance is altered by an inadequate intake, normal and abnormal external losses, and internal translocations of fluid and electrolytes, these three categories will be discussed separately.

Inadequate Intake—The oral intake of fluid and electrolytes may be inadequate in an adult patient who has psychiatric difficulties or in other individuals who are not willing or not permitted to consume sufficient amounts of nutrients. Although patients may have a reduced intake of food and fluid their caloric needs are not significantly decreased for several days thus they obtain energy by metabolizing fat and protein from their body tissues. Under these circumstances the insensible fluid and electrolyte losses continue and may even be accelerated if there is a fever and/or respiratory abnormalities. The kidneys may aid in the conservation of water and elec-

NAME:_____ DIVISION:_____ HOSPITAL NO. _____

AGE:_____ YRS. HEIGHT:_____ WT.:_____ KG. DIAGNOSIS:_____

	SOURCE OF LOSS	APPROXIMATE LOSS VOLUME ML.	mEq/L. NA	K	CL	1. H_2O	NA	K	CL	2. H_2O	NA	K	CL
BASIC	LUNGS	400-1600	0	0	0								
	SWEAT	400-6000	40	5	30								
	URINE	0-3000	2-200 mEq										
GASTRO INTESTINAL	GASTRIC	100-4000	60	10	130								
	BILE	50-800	145	5	85								
	PANCREAS	100-800	140	5	75								
	DUODENUM	100-2000	140	5	80								
	DUODENUM WITH GASTRIC	100-3000	135	5	100								
	ILEUM	100-9000	140	5	100								
	COLON	100-9000	80	50	40								
	TRANSUDATES AND EXUDATES	100-9000	140	4	100								
	TOTAL												
	BLOOD	500-5000	REPLACE VOL FOR VOL.			_____ ML.				_____ ML.			
	CALORIES (INCLUDING N)	KG. WT. _____ X 30 = _____ APPROXIMATE DAILY CALORIC NEEDS											
	NITROGEN WT X 0.5 X 6.25 = DAILY PROTEIN NEEDS	KG. WT. _____ X 0.3 = _____ APPROXIMATE DAILY NITROGEN NEEDS											
	VITAMINS	2 TO 10 TIMES MINIMUM DAILY REQUIREMENTS											

Table 1. Work sheet for calculation and tabulation of water and electrolyte requirements.

trolytes but often following an injury or during an illness the large amounts of nitrogenous waste products and acids which must be eliminated via the kidneys induce an obligatory loss of water. It therefore is necessary for the physician to supply the patient with the basic needs of water and electrolytes during many illnesses and following certain operations and injuries.

External Losses—Normally approximately 1500 to 2500 ml. of sensible and insensible fluid are lost each day. The external losses of fluids and electrolytes may be greatly increased in certain conditions such as excessive perspiration, vomiting, diarrhea, and drainage from wounds or from tubes in the gastrointestinal tract and from fistulae. It should be remembered that some times very large volumes of perspiration may be lost. There are instances on record where from 2 to 10 liters of fluid are lost from this source per day. The daily losses and concentrations of electrolytes in the gastrointestinal fluids which on occasions may also be lost externally are shown in Figure 2.

Although the chemical make-up of these fluids may vary the concentration of the electrolytes contained in the perspiration, saliva, and gastrointestinal fluids are approximately equal to the values shown in

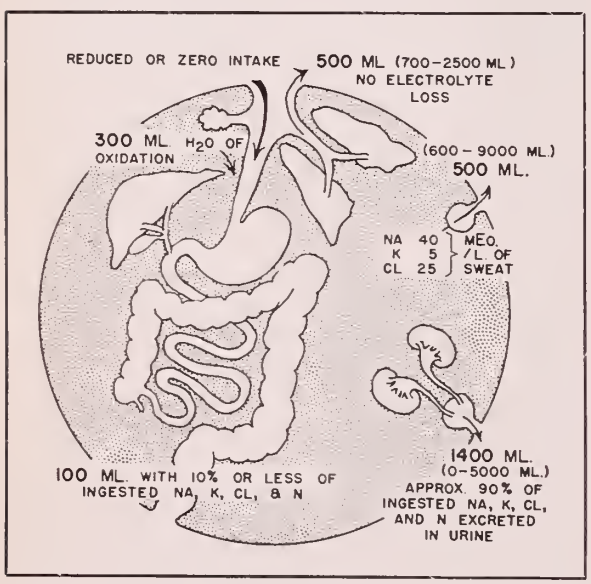


Figure 2. Gastrointestinal fluid and electrolytes which may be lost externally.

Figure 2. The concentrations of electrolytes in sweat and saliva are considerably lower than in the extracellular fluid. It also should be noted that the concentration of potassium is higher in saliva than in many of the other fluids and that there are no electrolytes present in respiratory water. In many instances mixtures of gastric and duodenal secretions as well as fluid from the lower small intestine and translocated fluids have a concentration of electrolytes comparable to that of extracellular fluid. Therefore considerable quantities of these types of fluids can be lost without producing a significant change in the plasma or serum electrolyte concentrations. When a patient vomits profusely or has lost fluid by drainage from the upper gastrointestinal tract, the kidneys will frequently excrete large amounts of sodium and potassium in order to compenstate for the excessive chloride loss in gastric fluid. In this way the acid-base balance or pH is maintained in spite of increasing dehydration and relatively normal serum electrolyte concentrations may exist. It is therefore necessary to provide both cations and anions in treating patients who have lost both fluid and electrolytes. When chloride is lost in the vomitus or drainage the patient should be given both sodium and potassium chloride. When there are losses of bile and pancreatic juice, which may contain large concentrations of bicarbonate, it is important to prevent hyperchloremic acidosis by the administration of sodium or potassium bicarbonate, citrate, gluconate or lactate. In patients with pyloric obstruction due to a peptic ulcer or in other individuals who lose large amounts of free hydrochloric acid by vomiting or gastric tube suction, the need for large amounts of chloride in conjunction with smaller amounts of both potassium and sodium should be obvious. Not frequently patients with peptic ulceration will show gastric fluid sodium concentrations which range from 30 to 60 mEq. per liter and at the same time because of a large amount of free hydrochloric acid will have a gastric fluid chloride level ranging between 125 and 150 mEq. per liter. It should be remembered that the primary source for large external losses of potassium is through the kidneys. Since the sodium concentration of lower smaller intestinal secretions

and of transudates and exudates is similar to that of the plasma, or extracellular fluid, the external loss of such fluids may greatly reduce the circulating plasma volume. The need for fluid with a chemical composition comparable to that of interstitial fluid in conjunction with a small amount of free water may be determined from the history, blood pressure, pulse, rate of urine flow and examination of the patient. It may frequently not be apparent from the laboratory findings alone.

Internal Shifts of the Body Water and Electrolytes (Formations of a Third Body Fluid Space)—Internal shifts of water and electrolytes occur following injury, infection, partial or complete obstruction (mechanical or neurogenic) of the gastrointestinal tract or venous circulation. These shifts may also occur when there are abnormalities in endocrine function, and in patients with cardiac, renal or hepatic disease. Maintenance or increase of body weight in a patient who has an inadequate caloric and protein intake indicates that he has retained significant quantities of fluid and electrolytes (third space).

Frequently shifts of fluid and electrolytes can occur quite rapidly so that an individual who appears well can be hypotensive and oliguric within a very short period of time. The rapidity and extent of this translocation of fluid into the "third space" is demonstrated by the case history of a twenty-six year old patient who was admitted to the University Hospitals of Cleveland several years ago. Five hours prior to admission he was in excellent health, having worked daily and had no interference with his normal food and fluid intake. He was awakened by severe intermittent cramping abdominal pains at about 3:00 a.m., vomited approximately 250 ml. of bile stained fluid, and shortly afterward came to the hospital where he again vomited a small amount of fluid. His temperature was slightly subnormal, pulse 120, blood pressure 82/50 and white count 16,500. The patient passed only a small amount of urine with a specific gravity of 1.028 and containing a slight trace of albumin. Because of small urine volume he was catheterized and no residual urine was found in the bladder. The patient was then given a 5 per cent dextrose solution in saline because he had been markedly oliguric for several hours, and this was followed by a 10 per cent dextrose solution in water to which small quantities of sodium lactate had been added. The presumptive diagnosis was a strangulated intestinal obstruction and a laparotomy was carried out. A volvulus of the small intestine around an inflammed Meckel's diverticulum was found. Approximately 700 ml. of serosanguineous fluid was removed from the peritoneal cavity. Because of the marked distention of the small intestine decompression was carried out by inserting a sump metal suction tube into the lower jejuneum near the junction of the ileum just above the point of obstruction. Over four liters of fluid was removed from the small bowel and it was noted that considerably more fluid was present in the intestinal wall, mesentery, and in the upper gastrointestinal tract proximal to the point which could be decompressed. It was believed that this patient had translocated and lost externally somewhere between 9 and 10 liters of electrolyte containing fluid in a period of about 12 hours. The intraperitoneal and intestinal fluid and that present as edema contained approximately 136 mEq. of sodium, 4 mEq. of potassium and 100 mEq. of chloride per liter and was equivalent to 7 to 8 liters of extracellular fluid. A total of 10,000 ml. of fluid was given to the patient from the time of admission until two hours postoperatively. This consisted of 1500 ml. of whole blood, 2,000 ml. of a 10 per cent dextrose solution and the remainder a 5 per cent dextrose solution which contained sufficient sodium chloride and sixth molar sodium lactate to provide about 135 mEq. of sodium per liter of fluid. His pulse rate decreased, blood pressure stabilized at a normal level, and urine flow increased after this quantity of water and electrolytes was given.

Figures 3, 4, and 5 depict schematically situations in which there may be a significant translocation of fluid and electrolytes from the normal body compartments. These are peritonitis, closed loop obstruction (sigmoid volvulus) and the shift of water and electrolytes into the small intestine during

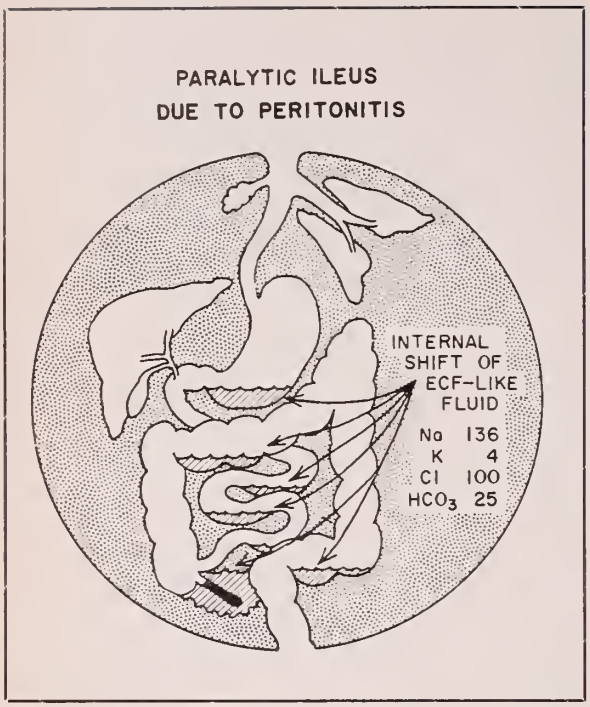


Figure 3. Internal shifts of fluid and electrolytes associated with peritonitis.

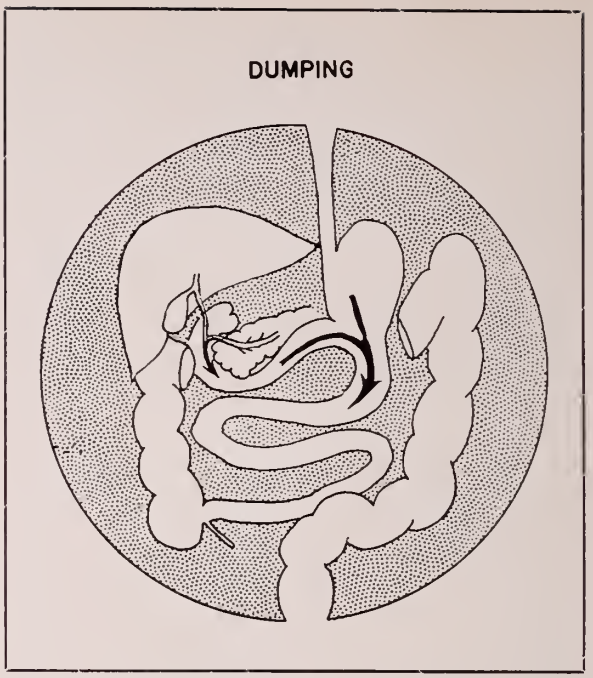


Figure 5. The internal shift of abnormal amounts of water and electrolytes which occurs during dumping.

Thus by estimating the magnitude of the patient's losses and sequestrations of fluid and electrolytes and at the same time considering the amounts of these materials required to meet the deficits plus the basic needs (insensible losses and urine formation) one can readily calculate the quantity a "dumping" attack following a gastric operation.

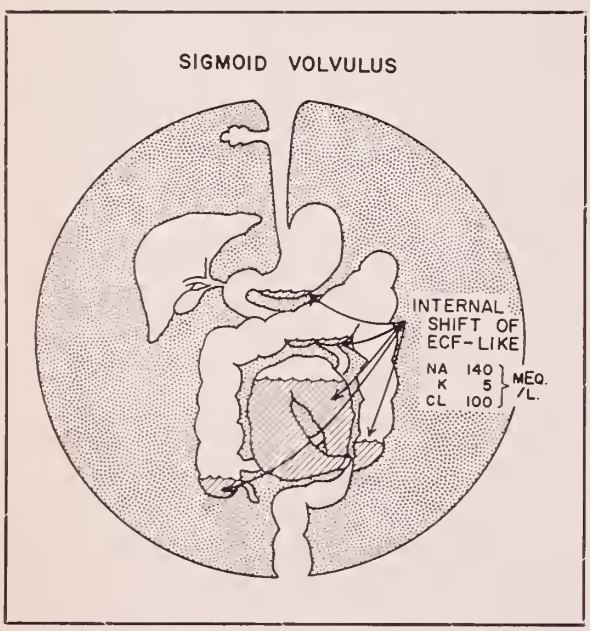


Figure 4. Third space formation (internal shift of water and electrolytes) associated with a sigmoid volvulus.

and composition of replacement needed. Using a scheme similar to that presented in Table 1, one can tabulate all losses and readily calculate the amount of replacement fluids. While the amount of fluid and electrolytes lost by external drainages or vomitus can be quite accurately measured, it is more difficult to determine the losses resulting from oozing of denuded surfaces or the magnitude of the third space. However, by periodic evaluation of the patient's response to therapy (thirst, eye ball tension, skin turgor, pulse rate, venous and arterial pressure and the rate of urine flow) it is usually possible to provide any necessary adjustments in intake. With this method of treating fluid and electrolyte aberrations one uses the concepts of simple chemical bookkeeping (balancing total losses against intake) rather than depending completely on complex laboratory procedures.

DISCUSSION

DOCTOR DuVAL. Associate Professor of Surgery, University of Oklahoma School of Medicine: Doctor

Abbott, have you found the sodium content of pancreatic juice to be fairly constant or would you advise measuring this concentration at intervals?

DOCTOR ABBOTT: We haven't had the opportunity to measure the sodium concentration in pancreatic juice too often. We did have a patient with a pure pancreatic fistula not long ago, and for the most part, our results showed that the sodium concentration is near isotonic with the extracellular sodium concentration. Others have also shown that following stimulation of the pancreas the chloride concentration of the pancreatic fluid drops to well below that normally found in plasma, while the bicarbonate concentration may rise from around 30 mEq/liter to a concentration which is two or three times as high as that in the resting gland. I'm not sure how the electrolyte concentrations are affected by disease. It would be very interesting to collect some of the pancreatic juice that Doctor DuVal and others obtain following pancreatic drainage procedures in patients with chronic pancreatitis.

DOCTOR DuVAL: In such studies as you have done have you found that there seems to be a discrepancy between your findings on volume output of pancreatic juice per day as compared to what is being currently reported in textbooks?

DOCTOR ABBOTT: Yes, I believe there is quite a difference. I think we were all taught that the normal individual secretes about eight to ten liters of fluid into the gastrointestinal tract each day, and I am sure, as Doctor John Peters emphasized many years ago, that it takes food and water to stimulate gastrointestinal secretions; and therefore, if little or no food and water are consumed, the volume of digestive juices would be greatly reduced. As soon as patients are taken off of everything by mouth, our studies indicated that the output of saliva, gastric secretions, bile, pancreatic juices, and succus entericus are greatly diminished. More recently we have been removing gastric suction tubes in 12 to 24 hours from postgastrectomized patients, keeping them off of everything by mouth, and periodically measuring any retained fluid if it sems necessary. We have been surprised to find very little fluid. I believe that it is important to remember that the amount of gastrointestinal fluid secreted, including pancreatic juice, fluctuates a great deal. Usually the secretion is more plentiful following the ingestion of food or water than that occurring in the patient who is not eating or drinking.

DOCTOR DuVAL: A question that has been posed on several occasions involves the preparation of a patient for surgery who has pyloric obstruction. I'd like to know if the losses are substantially different with constant suction, on such a patient, as opposed to letting the patient take fluid by mouth and emptying the stomach once or twice daily? Is there a difference in the losses with those two ranges of management?

DOCTOR ABBOTT: Yes I think there is considerable difference. Recently, we had a patient who insisted upon treating herself. We let her manage her oral intake while intermittently her stomach was emptied by a gastric tube. In this patient, we were able to make some observations of this type. When a person takes food and water, they secrete more digestive juices into the gastrointestinal tract. It's not really the water that the patient ingests that you worry about losing by suction, but the water and electrolytes that the patient secretes in response to the water they swallow. We don't believe that there is ever a need for an emergency operation in patients with pyloric obstruction, they should all be well hydrated and the acid-base balance and nutritional deficits should be corrected or restored toward normal before an operation is undertaken.

DOCTOR DuVAL: Would you discuss the problem associated with the electrolytes that have been badly deranged and then returned to normal, at which time the patient dies.

DOCTOR ABBOTT: I am glad that you touched on this problem, since the point I would like to make, and the point which I tried to make during my talk, is that a plasma or serum electrolyte concentration per cc measures only one dimension. It represents only concentration and gives no indication as to the total quantity. Obviously, we need a three-dimensional view, volume plus concentration, to be accurate. As McIver and Gamble emphasized so well in an article published in the Journal of the American Medical Association in 1927, if one lined up ten liters of saline solution and took a sample of fluid from any one of these bottles, the concentration would be the same. However, if somebody came along and knocked over and broke three of these liter bottles, the concentration in the remaining seven liters would still remain the same, although the total amount of water, sodium, and chloride has been decreased by 30 per cent. In a sense, this is what is happening in a patient after a thermal burn and in a patient with internal shifts into the peritoneal cavity and the other regions. He is losing fluid into a traumatized or inflamed area which has an electrolyte concentration exactly the same as that of plasma. Unless he starts sweating and breathing fast and losing water, which would tend to raise his serum electrolyte concentration, or unless we pour a lot of fluid which is low or free of electrolytes into him, which will lower the plasma electrolyte concentrations, his electrolyte values will remain at a relatively normal concentration. It is very disturbing to have somebody say that they have followed a patient who came to autopsy and he was in good fluid and electrolyte balance, since his serum concentrations were essentially normal. Yet, if you check the patient's intake or output, it becomes obvious that this aspect has been overlooked. It should therefore be remembered that balance of water or electrolytes can be determined only after careful intake and output studies and need not have any relation to the serum electrolyte concentrations.

A Practical Guide to Fluid Therapy in Burns

CURTIS P. ARTZ

THE PRIMARY DERANGEMENT after burn injury is loss of fluid from the intravascular compartment brought about by vaso-dilatation and increased capillary permeability. This loss of fluid leads to a diminution of the circulating blood volume and thereby the threat of so-called "burn shock." The amount of fluid lost varies with the extensiveness of the burn. Some fluid passes into the interstitial space, as is evidenced by edema formation. Additional fluid is lost as exudate into blisters and from the weeping surface of partial thickness burns.

Proteins, electrolytes and water pass from the plasma into the interstitial space immediately after the burn (Figure 1). Colloid, electrolyte solution and water must be given to maintain an adequate circulating blood volume and thus prevent hypotension. Some of this replacement therapy passes through the damaged capillary walls into the interstitial space, giving the patient an expanded interstitial space and thereby a combined load of water and sodium (Figure 2).

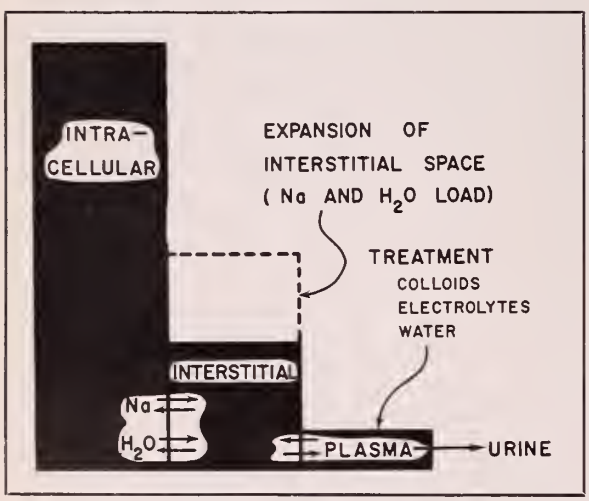


Figure 1. Diagram of body water compartment changes after burns. Soon after the injury plasma passes into the interstitial space. Treatment with colloid, electrolytes and water maintain the plasma volume but water and sodium continue to flow into the interstitial space. This expands the interstitial space as is shown by the dotted line. The increased amount of sodium and water in this interstitial space means that the patient has a combined load of sodium and water. This remains until redistribution of fluid occurs. There is an interchange of sodium and water between the intracellular compartment and the interstitial space, but this is not well defined. After the third day the interstitial space decreases in size and water is usually excreted faster than sodium.

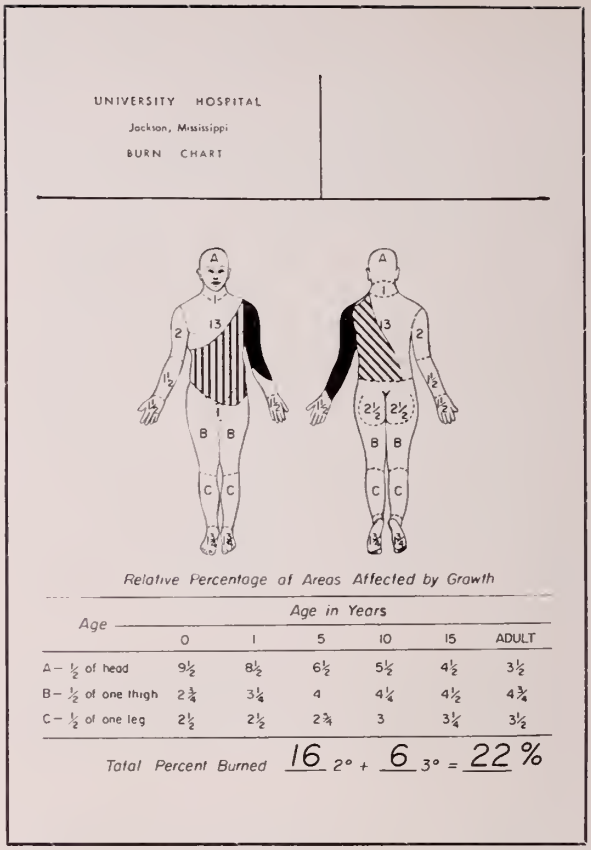


Figure 2. Typical hospital sheet with Lund and Browder chart. On this sheet is sketched the second degree (vertical lines) and third degree (solid black) areas of burn injury. This is the most accurate method of determining percentage of body surface burned.

This load slowly increases as treatment is being given until about 48 hours after the burn. At that time capillary permeability diminishes and fluid redistribution begins. Water and sodium are reabsorbed into the vascular system, and the interstititial space decreases. Excess sodium and water are excreted by the kidneys. In extensive burns it may take several days for this expanded interstititial space to return to normal.

The loss of red blood cells following burns has been studied extensively.[1,2] There is a loss of red blood cells after a deep burn because:

a) Many cells in the burned area are destroyed directly by heat.

b) Delayed hemolysis occurs for 24 to 48 hours after burning.

c) Thrombosis occurs in the capillaries beneath the burned tissue and causes a decrease in the red cell mass.

d) Many red blood cells are lost following sludging of blood.

From a clinical standpoint it can be assumed that 10% to 15% of the red cell mass is destroyed in any appreciable third degree burn.

The desirability in initial therapy is to provide enough colloid, electrolyte solution and water to maintain an adequate plasma volume without giving excessive replacement therapy that may overexpand the interstitial space.

Initial Evaluation

Unlike most patients who suffer shock, burned patients have predictable losses. The size of the patient and the extent of the burn permit an estimation of the amount and rate of fluid loss. The aim in initial management is to estimate this fluid loss and replace it as it is being lost, thereby preventing hypotension.

Several methods have been devised for estimating the extent or percentage of body surface burned. The most common method is the familiar "Rule of Nines."[3] This formula is easily remembered and very convenient, but it is not as accurate as the use of a Lund and Browder Chart (Figure 2).[4] Every hospital should have a Lund and Browder Chart on a record sheet available for use in the emergency room. By marking the burned area on this chart, one is able to estimate rather accurately the percentage of body surface burned.

At the same time that the percentage of burned area is being determined, an attempt should be made to estimate the relative amounts of second and third degree injury. Although the total volume of fluid required is about the same for second degree as for third degree injury, the type of fluid given is somewhat different. Patients who have primarily second degree burns can be adequately resuscitated with plasma, or a plasma volume expander, and electrolyte solutions. Patients who have appreciable amounts of third degree injury require additional whole blood.

Planning the Method of Intravenous Administration

Since the life of a burned patient depends on the infusion of replacement therapy, it is wise to plan initially the utilization of various routes of administration. In the extensively burned patient intravenous fluid is often required for ten to twelve days. During this time phlebitis and thrombosis may occur in several veins necessitating a change in infusion site from one vein to the other. Good veins for intravenous infusion may be difficult to find; therefore a plan for saving veins should be worked out before therapy is instituted.

As soon as the patient is admitted, a large gauge needle should be placed into any accessible vein and blood drawn for cross matching and a base-line hematocrit determination. An infusion of Dextran or lactated Ringer's (Hartmann's) solution should be started through the same needle. This will permit infusion of fluids until there is time to sit down and plan the steps in therapy.

If it is determined that prolonged intravenous therapy will be necessary, a cut-down must be performed. The cut-down cannula is inserted into a vein of any available extremity as distal as is practical. If thrombosis of the vein occurs, then the cannula can be moved proximally. If there is a burn of the upper portion of the body, the cut-down should be inserted into an ankle vein. If phlebitis occurs, this can be changed to the opposite ankle vein or an upper extremity vein if available. Should thrombosis occur in this area, a vein higher on the leg can be cannulated, and eventually a femoral catheter may be necessary. The use of a

percutaneous plastic femoral catheter is very advantageous; however, this method of intravenous administration should be saved as a last resort. If thrombosis occurs in the femoral or iliac vein, then the other veins of that extremity will not be suitable for use. The femoral catheter should not be left in place for more than seven days because of the fear of thrombosis and possibly embolism. In very extensively burned patients, particularly when all four extremities are injured, it may be necessary to give infusions initially by way of a femoral catheter. When this occurs, the catheter should be inserted in one side and allowed to remain for seven days, then moved to the other side.

Planning the Amount of Fluid Therapy

As soon as the initial procedures in the care of the patient and the assessment of injury are made, the amount and type of fluid therapy should be planned. Several factors influence the plan:

a) Extent and depth of burn

b) Weight of the patient

c) Presence of respiratory irritation

d) Age of patient

e) General physical status of the patient.

By the use of a fluid formula it is possible to estimate from the weight of the patient and the extent of body surface burned the approximate fluid requirements for the first 48 hours. In the patient who has inhaled noxious gases there is the danger of pulmonary edema, and therefore, somewhat less than the usual estimated amount of fluid therapy should be given. Likewise, elderly patients and patients with severe cardiovascular disease should be given minimal replacement therapy.

Usually intravenous fluids are required only in burns of more than 20% of the body surface. Occasionally, burns that are primarily full thickness or burns in children, although less than 20%, may require intravenous therapy. Most minor burns, however, can be treated by oral fluids. A salt and soda solution provides the most satisfactory method of replacement. This solution is prepared by dissolving 3 grams of salt (½ teaspoonful) and 1.5 grams of bicarbonate of soda (¼ teaspoonful) in a quart of water. This solution is tolerated better when given cold and flavored with lemon. It is a hypotonic solution with approximately 70 mEq. of sodium per liter. If vomiting occurs with oral electrolyte solution, then lactated Ringer's may be given intravenously in amounts of 2 cc. per kilogram for each per cent of burn.

In the more severely burned patient well planned and well regulated intravenous therapy is required. Many patients with such extensive burns that there is little chance of ultimate survival live for several days. If therapy is to be successful, advantage must be taken of the more or less predictable volume and rate of fluid losses. In this respect the physician has a therapeutic opportunity that is rarely encountered in other diseases characterized by disturbed water and electrolyte distribution. If judicious treatment is instituted within a few hours after injury, signs and symptoms of derangement can frequently be prevented. Because the volume and rate of fluid losses are predictable, several fluid formulas for estimating the amount and type of therapy have been devised. All seem to have merit. The Brooke formula has proved to be most successful in a very extensive experience.[5] This formula calls for the following fluids in the first 24 hours after injury:

a) Colloid (blood, Dextran or plasma)—0.5 cc. per kilogram for each per cent of body surface burned

b) Electrolyte solution (lactated Ringer's)—1.5 cc. per kilogram for each per cent of body surface burned

c) Water requirement (dextrose in water) Adults—2,000 cc.

Water requirements in children vary. The

following is a rough guide to daily water requirements in infants and children:

a) During the first two years—120 cc. per kilogram

b) Second to fifth year—100 cc. per kilogram

c) Fifth to eighth year—80 cc. per kilogram

d) Eighth to twelfth year—50 cc. per kilogram.

Burns of more than 50% of the body surface should be calculated as 50% burns.

The above formula is only an *estimate* of fluid requirements, which allows a *plan of therapy*. The exact amounts of fluids given should be in accordance with the response of the patient. It is usually anticipated that ½ of the above fluid requirements should be given in the first 8 hours, ¼ in the second 8 hours, and ¼ in the third 8 hours. This rate of administration is based on the fact that fluid losses early after the burn are more rapid than later.

The depth of burn influences the type of fluid therapy, particularly in the colloid requirement. Second degree burns up to 40 per cent of the body surface will do well if the colloid requirement is met by the use of Dextran or plasma. Burns that have an appreciable amount of third degree injury should be given the colloid requirement as half Dextran and half whole blood. The protein content of plasma would favor it as a therapeutic tool; however, it is considerably more expensive and virus-free plasma is not a readily available as is Dextran. Most clinicians, therefore, prefer Dextran to plasma as initial colloid therapy.

Physiologic saline solution is used extensively as an electrolyte requirement. It is inexpensive and readily available. Its chief drawback is the excessive high concentration of the chloride ion. Burned patients have a tendency toward metabolic acidosis, and massive infusions of physiologic saline solution are followed by hyperchloremia, which tends to enhance the acidosis. A balanced electrolyte solution, such as lactated Ringer's, is more acceptable. When lactated Ringer's is not available, a satisfactory electrolyte solution containing 159 mEq. of sodium and 103 mEq. of chloride per liter can be prepared by mixing physiologic saline solution and 1/6 molar lactate solution in proportions of two volumes of saline to one volume of 1/6 molar lactate.

In some patients the water requirement may be given orally. Most severely burned patients, however, develop a paralytic ileus early, and vomiting may result. It is usually safer to withhold all fluids in a seriously burned patient and give the entire water requirement during the first 24 or 48 hours by the intravenous route.

As soon as the plan of fluid therapy has been determined, it should be written down, but it is expected that it will be changed. As Doctor Francis Moore has so aptly put it, "Planning fluid therapy in the early burn is like a family budget. It is made to be broken."[6] As the plan is followed, the actual amount administered is dictated by the clinical response of the patient. It is foolhardy to think that fluids can be estimated and ordered on a 24 hour basis. There is no substitute for hourly observation. The type of therapy and rate of infusion must be changed frequently in accordance with the patient's response.

Following the Plan of Therapy

To follow the fluid therapy adequately in a burn of more than 25% of the body surface, it is absolutely manditory that an indwelling catheter be placed in the bladder so that the hourly output of urine can be measured. It should be removed, however, as soon as possible, or urinary tract infection may develop. After the initial cutdown has been performed, and an adequate amout of plasma volume expander, blood, or lactated Ringer's has been given, attention should be directed toward local care. Anesthesia is unnecessary and contraindicated. As soon as the local burned wound

has been treated and the patient has been taken to the ward, a plan for following fluid therapy should be outlined. If several physicians are assisting with the care of the patient, one should be delegated as having the primary responsibility of writing orders and determining the speed and type of fluid administration. A chart should be prepared to record hourly urinary output, blood pressure, and intravenous infusions.

In general most of the colloid requirements should be given early. This will help in maintaining an adequate plasma volume and prevent hypotension. If the patient has had no fluid therapy for several hours between the time he is burned and the time of admission, very energetic colloid and fluid therapy may be necessary. In very severe burns there may be no urinary output for a few hours. During this time energetic colloid and electrolyte therapy should be given. Once the urine has started to flow, it can be assumed that the intake is catching up with the losses from the vascular tree. After observing the urinary output for a few hours, a good estimate of the rate of infusion can be made.

The chief value of the estimated fluid requirements by the use of a formula is that it allows knowledge of the ratio of colloid to electrolytes to dextrose and water. As the response of the burned patient is determined, a fall in blood pressure with fall in urinary output means additional colloid therapy. A fall in urinary output with a stable blood pressure means that additional electrolyte solution or water is required.

The patient's response must be re-evaluated every hour or two and fluid intake regulated. After 8 to 12 hours it may be possible to predict what will be necessary in the way of intake, and observation then can be less frequent.

One of the great dangers that occur in early fluid therapy of burns is excessive intake. It is not uncommon for a physician to assume that 30 cc. per hour urinary output is good, but 50 to 100 cc. is better. This is a dangerous fallacy. The aim in early fluid administration is to give the least possible amount that will assure a plasma volume sufficient to maintain a pressure in the glomeruli adequate for the output of urine. Additional fluid therapy leads only to added expansion of the interstitial space. Excess water and electrolytes in amounts that will produce a urinary output of 50 cc. or more per hour will lead to overexpansion of the interstitial space. This means an increased load of sodium and water. A urinary output of 15 cc. per hour is an indication for more rapid therapy, and an output of 50 cc. per hour should be followed by a reduction in rate of administration.

In infants, elderly patients, and patients with hypertensive vascular disease or irritation of the respiratory tract, the urinary output should be kept at 10 to 15 cc. per hour. In modern day treatment of burns excessive fluid therapy is a more common error than inadequate fluid therapy. Restlessness is usually a sign of inadequate therapy. In almost all burned patients there is a tremendous thirst which disappears in the well hydrated patient in 12-18 hours.

At times when the blood pressure is normal, additional colloid therapy may not result in a satisfactory output of urine. When such an event occurs, colloid therapy should be discontinued and electrolyte solutions of water given. By following the urinary output and the blood pressure, one is able to determine whether the change in urinary output is due to a lack of fluid, specifically water, or due to a diminished plasma volume with an inadequate flow of the renal glomeruli.

In patients with burns of the head occasional cerebral edema may occur during the course of treatment. This phenomenon is more common in children. To prevent this disastrous complication it is necessary to follow the temperatures carefully during the first 48 hours. An unusual temperature rise to 104° in a well hydrated patient may mean the beginning of cerebral edema. The eye grounds should be observed, and if there is any evidence of edema, the infusion of fluids should be decreased immediately. The mechanism of formation of cerebral

edema in burned paients has not been explained.

During the second 24 hours, the patient will require the same amount of water and about one-half the colloid and electrolyte requirement of the first 24 hours. Laboratory determinations during this period are of little value and are unnecessary. During the second 24 hour period, the ratio of colloid, electrolyte solution, and water should be calculated and administered according to the urinary output.

Therapy After Forty-Eight Hours

At 48 hours wound edema is maximal and at this time fluid redistribution occurs. Usually water and sodium return to the vascular system and the urinary output increases. In moderate burns the patient will regulate his fluid needs by oral intake. In more extensive burns some of the fluid requirement may be taken orally and the remainder intravenously in accordance with the urinary output and the serum sodium concentration. In the very extensively burned patient it may be necessary to continue fluid by the intravenous route for several days.

After 48 hours the laboratory may be quite helpful in determining therapy. Careful observation should be made of the hematocrit and the serum sodium. If the hematocrit falls below 45, blood should be given. Hyponatremia is usually present in spite of a large sodium load in the interstitial space. A low serum sodium in injured patients, particularly in burn patients, is associated with a good clinical response and is desirable. The insensible water loss may be quite marked, up to 5000 cc. a day in an adult. After the acute stress of the burn injury the body has a tendency to hold on to sodium, and very little is excreted by the kidneys. The loss of water by the insensible route and by the urine may be considerably greater than the loss of sodium. This means that the water load is diminishing more rapidly than the sodium load, and therefore a rise in serum sodium may occur. A serum sodium of 145 mEq. per liter or more is an indication for additional water. When insufficient water is given, the serum sodium may give rise to a hypernatremia.[7] The increased concentration of sodium in the interstitial space leads to intracellular dehydration and sometimes death. Daily serum sodium determinations, therefore, are very important in the extensively burned patient, and water should be given to prevent hypernatremia.

After the first 72 hours potassium may be required. When the patient can take nourishing fluids and food orally, intravenous potassium therapy is unnecessary. In the more severely injured patient it may be necessary to give 80 mEq. of potassium a day by the intravenous route until the potassium requirement can be met by oral intake. Later in the postburn period intravenous therapy is required to maintain an adequate red cell mass and in some instances for additional protein therapy. When the patient is unable to take adequate proteins and calories orally, some type of intravenous protein, such as protein hydrolysate or plasma, may be necessary as a supplement. The hematocrit should be determined twice weekly, and an adequate amount of blood given to maintain a hematocrit of 45 or above in the well hydrated patient.

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DISCUSSION

DOCTOR DuVAL: Doctor Artz, with respect to Dextran, I'd like to ask two questions: What is the situation as you know it with respect to prolonged bleeding time associated with the use of Dextran? What do you currently recommend as the best plasma volume expander for standard use in an emergency situation in a small hospital?

DOCTOR ARTZ: There appears to be an increased bleeding tendency in some patients with large quantities, two or more liters, of Dextran. This change in bleeding time does not become clinically significant by increasing the amount of bleeding after injury unless a large quantity of Dextran is given. This does not occur in all patients. I have seen some patients bleed rather profusely after giving them about two liters of Dextran. On the other hand, I have seen many patients receive two and one-half liters of Dextran with no increase in bleeding. I believe that most people who use Dextran use only one liter or maybe a liter and a half. This certainly is safe, from a clinical standpoint.

Second, where do we stand with the plasma volume expanders? There may be some plasma volume expanders coming on the market that will be better than Dextran, but it is doubtful that this will occur for a long time. Plasma is still best, but it is difficult to obtain appreciable quantities in the virus-free form. It is also quite expensive. The gelatins have not been very effective.

I would say that Dextran is a fine plasma volume expander and should be used whenever a plasma volume expander is needed.

DOCTOR DuVAL: Doctor Artz, may I ask you quickly on behalf of at least a few people in the group, who I am sure would like to know, where can one obtain a copy of the Lund and Browder chart? Do you have a ready reference for this?

DOCTOR ARTZ: In two places: (1) Lund, C. C. and Browder, H. C. The Estimation of Areas Burned. Sug., Gynec., and Obst. **79:** 352, 1944, (2) Artz, C. P. and Reiss, E. The Treatment of Burns. Philadelphia, W. B. Saunders Co., 1957, p. 10.

DOCTOR DuVAL: May I ask you the question that has been posed by several people. The so-called third space in burns, unlike the third space as seen in bowel obstruction where we are removing fluid physically at one point or another during the course of therapy, brings up the question of the danger of treating a deficiency secondary to this translocation of the fluid. As the patient recovers and the translocated fluid returns to its usual locus, with overhydration and secondary hypervolemia, how do you determine that this is occuring? How do you manage the patient during this period?

DOCTOR ARTZ: First, I think that all injured patients and all people with difficulties in the gastrointestinal tract not only have losses to the exterior, but losses into the peritoneal cavity, into the lumen of the bowel, into the bowel wall, and into the wall of the mesentery. This gives a **load** of sodium and a **load** of water. When fluid redistribution occurs, there is usually an increase in the urinary output. With this increase in output, the best method of measuring whether or not adequate amounts of water are being given is by determining the serum sodium. In other words, you choose not to over-hydrate the patient during this period. If his urinary output begins to increase, then you hope this is redistribution of the fluid. The only real worry is that he be given an adequate amount of water for insensible water loss. One looks at the patient, his surroundings, his fever, the temperature of the room, and tries to estimate the amount of water to replace his insensible losses. Then, if the serum sodium rises rapidly, electrolyte-free water must be given. The important thing is to estimate the insensible loss and try to replace it and not try to use the urinary output as regards to how much therapy should be given.

DOCTOR DuVAL: How about using marrow infusions? And while on that, you might just briefly state whether or not you feel there is any contraindication in using the same extremity as may be involved with the burn.

DOCTOR ARTZ: I'm not a real expert on sternal infusions of the bone marrow. A few years ago we had quite an experience for about a year attempting to infuse both the sternum and the tibia. For a few liters of fluid or for 500 milliliters of blood, it seemed to work well, but after that large volumes and quantities will not go in very well. At least, I've been unable to do it in my own experience. I think if one wants to give a unit of blood or maybe one or two liters of fluid and there are no other routes, that it is probably worth trying.

Secondly, I see no reason why one should not look down beneath the burned tissue for a vein. Sometimes if it is a full thickness burn, it is very difficult to find one. I think all of us have gone through burned tissue and inserted an indwelling polyethylene catheter. However, when one does this, you expect infection to go into the venous system and set up a bacterial as well as irritated type of thrombophlebitis, and this route does not last very long.

The Practice of Fluid Therapy

R. TARAIL

MANY OF OUR FAILURES in regard to fluid therapy are not the result of our failure to keep up with the latest advances. In a great many instances poor fluid therapy is little more than a failure of the physician, the nurse, and the hospital in quite another direction. Thus poor fluid therapy may occur because we don't activate long-established principles of good medical care as well as humane, diligent nursing and administrative practices. At the risk of belaboring what may seem to some to be a rather obvious point, I wish to emphasize this thesis further.

Please ask yourselves these questions. How often is the practice of fluid therapy impaired by inadequate history taking? How often is the practice jeopardized by hurried physical examination? How often is it given a black name by utterly inaccurate measurements of intake and output? How frequently do we neglect having accurate daily measurements of body weight? How often are our best intentions endangered by hospital chemical laboratories which lack professional supervision and which in many instances can be shown to be unreliable? How often is the successful practice of fluid therapy impaired by insufficient attention to underlying illnesses?

I shall try to concentrate our attention upon three commonplace aspects of fluid therapy: 1. Risks of air embolism; 2. The matter of how rapidly or slowly parenteral fluids should be given; and 3. The utility of accurate, daily measurement of body weight especially in bed fast patients.

Risks of Air Embolism

At least one catastrophe at our hospital pointed an accusing finger at the risks of cavalier attention to standard equipment used daily for giving blood transfusions. The familiar Y-type set (Figure 1) as emphasized by Kerr[2] is constructed with the built-in possibility of causing air embolism. Until Kerr's convincing demonstration of this possibility at our hospital I was ignorant of it. I am therefore bold enough to assume that this source of catastrophe has escaped the attention of many of you as well.

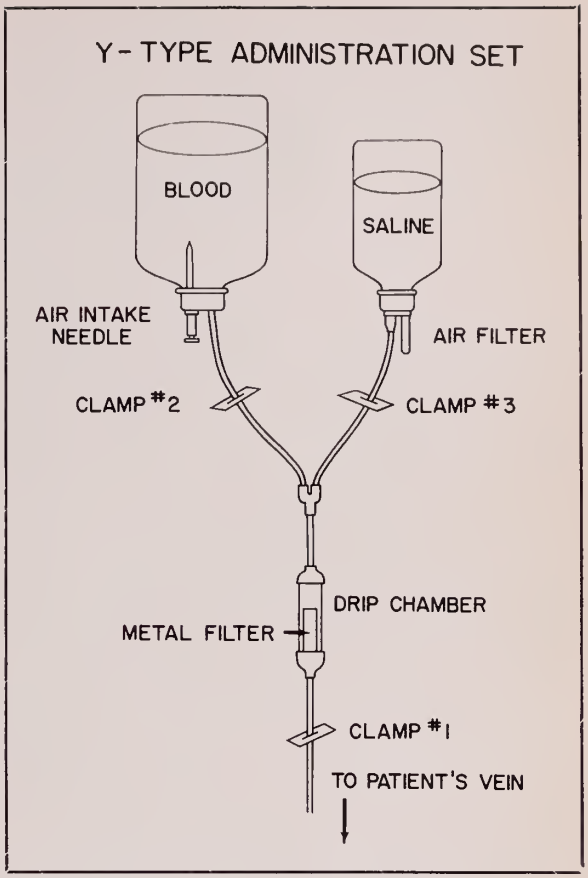


Figure 1. Practice of Fluid Therapy.

Assume for the moment that the Y-set picture in Figure 1 is intact and perfect in its manufacture and has no defects when used for transfusion. An infusion of blood is completed, and clamp No. 2 remains open, clamp No. 1 is completely open, and clamp No. 3 constricts the tubing and controls the rate of saline administration. *Under these circumstances large quantities of air can be shown to enter the tubing below the drip chamber at a rapid rate.* The source of this air is through the air-intake orifice of the empty blood bottle.

That this inrush of air occurs under the above-described circumstances is readily verified by anyone curious enough to spend a little time manipulating the controlling clamps. The explanation for this entry of air became clear to me through discussions

with a biophysicist at our hospital, Doctor Clinton Janney: It can be shown when the blood bottle is empty, clamp No. 1 and No. 2 open, and clamp No. 3 partially constricts the tubing so that the pressure in the lumen above clamp No. 3 exceeds atmospheric. In sharp contrast however the pressure in the lumen below constricting clamp No. 3 on the saline limb of the Y and below the junction with the blood side is distinctly less than atmospheric. These facts can be vividly verified by inserting a suitable needle into the lumen of the tubing above constricting clamp No. 3; this results in an outpouring of saline solution because the pressure within the lumen above clamp No. 3 is greater than atmospheric pressure. But if a needle is inserted into the lumen of the saline limb of the Y below constricting clamp No. 3, numerous bubbles of air can be seen to enter because the pressure within the lumen below clamp No. 3 is less than atmospheric. Air may enter from the empty blood limb of the Y because when clamp No. 2 remains open the pressure within the lumen of this limb is atmospheric (freely communicating through air intake needle) whereas the pressure at the junction of the Y (below constricting clamp No. 3) is less than atmospheric.

A special, perhaps less obvious, example of the same conditions develops if the blood bottle is empty, clamp No. 2 is open, and clamp No. 3 constricts the tubing to a greater extent than clamp No. 1. Again, large quantities of air may enter the tubing below the drip chamber rapidly and a similar explanation obtains.

A moral of this story is that after the blood has run in clamp No. 2 must obstruct the lumen completely, clamp No. 3 should be opened completely, and clamp No. 1 should constrict the lumen and control the infusion rate. Unfortunately the clamps of leading manufacturers do not readily lend themselves to easy assessment of just how much they constrict the lumen. All or none clamps, such as a spring clamp or hemostat at the upper limbs of the Y are far superior, and much less dangerous. And, incidentally, instead of the throw-away clamps provided at position No. 1 a well-made tunnel or screw-type clamp is desirable for rate control. Furthermore since, in a sense, an empty blood limb of the Y is precisely analogous to a needle poised to puncture the tubing, serious consideration of the advisability of abandoning Y sets appears to be in order.

Let us turn now to some of the risks of air embolism related to the use of defective sets for fluid administration. Holes may be present in the plastic tubing as a consequence of faulty manufacture, careless handling, or needle punctures. If the drip chamber and plastic tubing are fabricated as separate components there is danger of a leaky junction. Such defects may occur not only in Y sets but in single channel sets as well.

It can be readily demonstrated that the position of the constricting clamp controlling the rate of fluid flow is of critical importance. For example, consider a single channel set with a single fluid control clamp. If the tubing is punctured above the constricting clamp fluid leaks out. In contrast, if the puncture is below the constricting clamp large quantities of air may enter the tubing and travel to the vein.

The constricting clamp controlling flow should, therefore, be as close to the vein used for injection as possible. The fluid administration set should be tested for leaks prior to use.

The above discussion, admittedly incomplete, is intended to awaken interest (and vigilance) in air embolism as a significant hazard of fluid therapy. Additional information can be found in reviews by Kerr[2] and by Bailey.[1]

Rates of Fluid Administration

"Morphine Sulfate 15 mg. (h) p.r.n." If this order were to appear in the physician's order book of a well-run nursing station, the nurse would probably insist on a statement of how frequently the morphine may be given. Otherwise the order as erroneously written permits a rate of morphine injection of, for example, 15 mg. every five minutes.

"One liter of 10% glucose in isotonic saline i.v." Unfortunately this is the customary form of an order for intravenous fluids and, in this instance, the nurse will probably *not* insist on a statement of injection rate. Nevertheless under certain circumstances the intravenous injection of fluids at an improper rate may be as catastrophic as overly rapid administration of morphine.

A physician's order for parenteral fluids which fails to specify the desired rate of flow both in drops per minute and in c.c. per hour is written incorrectly. It should be a hospital rule that the fluid prescription includes both of these rate specifications. If these rate factors are omitted, or if they are included but not conscientiously enforced, the rate of fluid administration becomes a guess work affair inviting the serious consequences of too rapid or of too slow infusion. Why most of us consider it the physician's responsibility to decide upon the quantity and composition of intravenous fluid but in effect frequently evade the responsibility of selecting *and enforcing* an appropriate rate is something of a mystery.

It is surprising how often, in attempting to exercise control over rate, the physician assumes that one c.c. of fluid is equivalent to 15 drops. The number of drops in a given volume of fluid injected through an infusion drip chamber is determined primarily by the size of the orifice which delivers the fluid into the chamber. This orifice varies in size depending upon the manufacturer. For example, Abbott sets deliver about 20 drops per c.c. of fluid whereas Baxter sets deliver only 10 drops per c.c. Abbott has recently released a so-called microdrip affair which provides 43 to 45 drops per c.c. (useful for slow rates of flow; for example in babies or children).

The control of flow rate is critically related to the character of the clamp used in constricting the tubing. Many of the disposable sets provided by leading suppliers of parenteral fluids have clamps which require much time and effort to achieve precise adjustment of rate. Such clamps should be replaced by suitable screw or tunnel-type clamps. Nevertheless, not even the most precise clamp is a substitute for eternal vigilance since unless the infusion rate is checked and if necessary adjusted at frequent intervals, rates of flow may undergo striking increase or decrease as a result of change of position of the extremity, change in tone of the vein, or change in location of the needle.

Several types of disaster can be traced to lack of interest in rate control. Thus blood and other fluids given too fast to patients with diseases of the heart or lungs have produced pulmonary edema. Fluid administered too slowly has failed to remedy dehydration and mineral deficits. Glucose and other hexoses injected too rapidly have resulted in self-defeating melituria, osmotic diuresis, and increased urinary loss of electrolyte. Infrequent observation of flow rate has caused "runaways;" this is particularly unforgivable and tragic in infants and children. When one delves into the causes of such errors it may be apparent that the prescribing physician had intended that the fluid be given at a proper rate. In this sense the catastrophe may be regarded as a failure of the physician to translate intention into actuality.

An atmosphere of cooperation and understanding must be cultivated in each hospital in regard to this matter of rate control. Thus nurses, housestaff, attendants, and attending physicians must come to appreciate the importance of enforcing prescribed rates of flow 24 hours per day and seven days per week. Their appreciation of this can come from checks on flow in the course of fluid therapy in a given patient as well as from conversations, lectures, demonstrations, memoranda, and ground rules. Adequate quality and quantity of personnel and facilities are obvious prerequisites. The practical aspects and plans are best analyzed on an individual basis taking into account the peculiarities of the individual hospital.

There are all too few rules of thumb which I can proffer in regard to exactly which flow rate should be selected for a given situation. Among usually recognized factors which must be taken into consideration are: Type and quantity of fluid prescribed; technical matters such as availability of veins; comfort of the patient; continuous preven-

tion of ketosis; status of heart, lungs, and kidneys; rates of glucose utilization. The latter is probably less than 0.5 grams of glucose per kilo of body weight per hour in the starved subject because of "starvation diabetes." In the hydrated, non-starved subject a safe figure is probably about 0.5 grams per kilo per hour or somewhat higher. It is very useful to test the urine for sugar at regular intervals. If glycosuria is less than 3+ it is probably of little importance in causing wastage of water and electrolytes.

Daily Measurement of Accurate Body Weight

How much fluid shall I prescribe for this patient during the next twenty-four hours? This is one of the burning questions each clinician strives to answer satisfactorily as he treats patients with parenteral fluids. A valuable help in answering this question is afforded by knowing whether the water stores of the patient have increased, decreased, or remained unchanged. A simple example is the patient who on day No. 1 is assumed to have a normal quantity of body water, and who 24 hours later is found to have sustained a net loss of body water approximating 2 liters. The crux of the matter is that estimates of change in the quantity of body water are best and most accurately made by serial (usually daily) measurement of body weight.

History, physical examination, chemical and other laboratory data, measurement of intake and output, knowledge of underlying illness—all are highly significant, invaluable contributors to successful fluid therapy. But, useful and crucial though they may be, none holds a candle to the utility of serial body weights in reflecting day-to-day changes in body water. And a bit of reflection should show that knowledge of change in body water is one of the principal determinants of the answer to the question: How much fluid is appropriate for the next 24 hour period?

It can be reasonably assumed, for purposes of fluid therapy, that about 0.1 to 0.4 kilograms of body weight is the range of daily loss in an adult sustained on parenteral fluids provided that no deficiency of body water has developed. The extent of this non-water component of weight change depends upon the balance between caloric intake and caloric need. This balance may vary considerably. But even if the caloric intake is negligible, non-water loss should fall within 0.4 kilograms if adequate fluids are provided. For example, if accurate body weights show that a patient has lost 0.3 kilograms of weight in 24 hours, an insignificant change in body water can be inferred. On the other hand, if the authentic 24 hour weight loss is 2.3 kilograms a decrease in body water of about 2 liters is suggested.

This estimate of change in body water is most readily inferred from change in weight rather than from other information which can be brought to bear on the problem. Thus, consider a patient who on day No. 1 is normally hydrated and weighs 60.0 kg. (Table 1). Assume that this patient is losing fluid from the gastrointestinal tract over the next few days and parenteral fluids are the exclusive source of intake. The daily loss in body weight shown (Table 1) is less than 0.4 kg. Therefore it can be safely assumed that no significant change in the total quantity of body water has occurred. Suppose, however, that the weight were found to be 57.7 kg. instead of 59.7 kg. on day No. 2. This loss of 2.3 kg. would define a deficit of body water of about 2 liters.

Day	Weight (Kg.)
1	60.0
2	59.7
3	59.7
4	59.4

Table 1

How readily can this 24 hour loss of 2 liters of body water be detected by means other than serial measurement of body weight? The clinical history and physical findings during this 24 hour period would at best yield useful qualitative information. Accurate measurement of intake and output is an indispensable component of optimal fluid therapy. Even such measurement, however, does not include variable

loss of water through skin and respiration. Nor are anything resembling accurate measurements of output of fluid usually available to the clinician. Moreover certain types of gastrointestinal losses (among others), for example profuse drainage from a fistula, vomiting, and diarrhea, lend themselves poorly to quantitative collection. Hence measurable intake and output are unworthy substitutes for serial body weights in relation to change in body water. As already emphasized, however, serial body weights do not yield the vital information gleaned from knowledge of intake and output. The latter data are useful in judging the composition of replacement fluids; body weight change sheds no light on this problem.

Laboratory data generally available in hospitals, although crucial for many aspects of fluid therapy, would yield no more than a clue concerning the 2 liter deficit of water. Such measurements would not quantitate the deficit to the extent derived from change in body weight unless direct, ordinarily unavailable, serial measurements of body water were undertaken.

The above reasoning establishes the supremacy of serial measurement of body weight in estimating change in body water and thus in helping to guide the quantity of fluid prescribed. At the same time body weights are not proper substitutes for clinical history, physical examination, laboratory data, and measurements of intake and outgo. These have their own special utility and, although beyond the scope of this presentation, all must be taken into account in devising a proper fluid prescription.

Two major problem appear to conspire against obtaining body weight measurements which are meaningful for fluid therapy: Firstly, the bed fast patient is frequently not weighed because of alleged technical difficulties. This fact is in a sense tantamount to stating that the more serious the illness the less is the need for objective criteria to guide fluid therapy. There is at least one commercially available scale[3] which facilitates weighing of bed fast patients. Secondly, many measurements of body weight are about as useful as no measurements at all. This is true because of the grim fact that body weights as ordinarily measured in hospitals are subject to gross inaccuracy. This then resembles the situation in regard to accurate intake and output data save for the fact that obtaining a precise body weight is intrinsically simpler and less demanding of time and effort.

Weighing a patient with a blanket on one day and without this blanket 24 hours later may be reported as a loss of body weight of 2 kg. or more. This apparent loss of body water could lead to disastrous treatment. Physicians charged with the responsibility of fluid therapy have by this very fact accepted the responsibility for providing a carefully thought-out, continuously supervised program of body weight measurements which meets the needs of their individual hospitals. Even those prescribing fluids for infants and children oftentimes ignore the comparatively simple problem of arranging for accurate body weights and evidently prefer instead a kind of roulette wheel guidance. Is it unreasonable to suggest that an era in which gargantuan feats in the operating room are almost commonplace should also be characterized by the ease with which accurate body weights can be obtained and interpreted?

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DISCUSSION

DOCTOR DuVAL: Doctor Tarail, would you restate the situation with respect to the drops per minute. It may have been implied this morning that the number of drops per minute varies with the tonicity and specific density of the solution. Although this is actually true, from a practical clinical viewpoint, I think you were probably referring more to the size of the nipple of the equipment itself. Would you like to restate that?

DOCTOR TARAIL: Yes. Doctor DuVal hit the nail on the head. It makes very little difference from the practical point of view what the specific gravity of the solution being administered is. Even blood, which has higher specific gravity, of course, than most solutions, can be used with the figures that I gave you. The size of the drop is determined by the characteristics of the equipment. With Baxter sets, irrespective of the type of fluid being administered,

one can, in round numbers, say that ten drops is equivalent to one milliliter of fluid; and with the Abbott set, with its size of opening, one has to allow for twenty drops per cc. of administered fluid. The specific gravity is then a relatively minor, and from a practical point of view, insignificant part of this problem.

DOCTOR DuVAL: In what position should a patient be placed, in whom air embolism is suspected, in order to prevent air embolism to the brain; and does the venous pressure of the patient have any bearing on the occurrence of air embolism?

DOCTOR TARAIL: It is generally advised that the head be quite low, below the rest of the body, so that bubbles will not rise to the head and injure the vital structures, but rather go into the vessels lower down. Then the bubbles can be reabsorbed in the course of time, relatively safely. In addition, the patient should be placed on the left side. This is thought to allow for an accumulation of the air in the apex of the right ventricle. Whereas, if the patient is lying flat, there is more tendency for accumulation of the air at the orifice of the pulmonary outlet from the right ventricle. If the bubbles are placed so that they are next to the orifice leading to the lung, then there will be an ineffectual contraction of the ventricle upon the air. The air is compressible, unlike the blood, and nothing will move. To recapitulate, the patient should be on the left side, with the right side up, and the head very low.

In regard to venous pressure, this, of course, is an important matter in the hydrostatics of the situation in the fluid therapy. If, for some reason, one has an infusion going into the jugular vein and the situation is such that during a phase of the respiratory cycle the venous pressure is below atmospheric, this, of course, will enhance any tendency there might be, because of defective equipment or defective use of equipment, for air to enter. Conversely, if a person has a normal or a high venous pressure, this will tend to oppose the inflow of air.

DOCTOR DuVAL: On the subject of formulas, there must be some here who are wondering, in considering the daily weight, do you use any correction factor or do you assume that there is a one-for-one ratio between weight loss and fluid loss?

DOCTOR TARAIL: One can use the figure 0.1 to 0.4 kilograms for body weight loss as the loss that accrues as a result of tissue destruction **sans** water. So that in regard to dehydration, if the loss of weight exceeds 0.4 kilograms, then one begins to pay heed to it. If it's within that range of 0 to 0.4 klios in 24 hours, this is to be considered as a result of tissue destruction, or weighing the patient with stool one day and without the next day.

And, of course, as Doctor Abbott so neatly pointed out in the course of his discourse, the body weight is no substitute for taking all of the information that can be brought to bear on the problem into account and thinking about it in a slow, meticulous, detailed way.

Use of Osmotic Diuretics in Surgery

J. U. SCHLEGEL

THE PHYSIOLOGICAL HANDLING of water by man is governed by some basic rules which should be appreciated before one can evaluate the changes resulting from trauma. This discussion will be limited to the altered handling of water and electrolytes that occurs following trauma in patients with normal kidneys.

It is generally not realized that a normal individual has a limitation in his ability to excrete water, which is directly related to his intake and excretion of solutes. Kerrigan, Talbot, and Crawford[1] have diagramatically expressed the normal range of physiologic tolerance for water (figure 1) based upon the ability of normal individuals to handle a water load under conditions of varied solute intake. A normal individual who receives only dextrose and water for a 24-hour period has a tolerance for water of

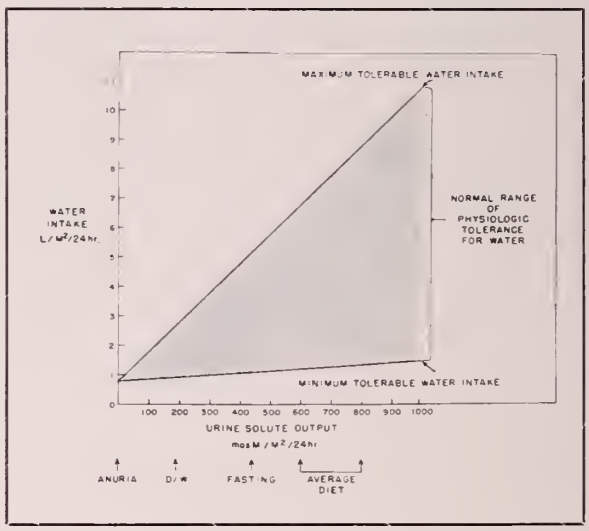


Figure 1. Influence of urine solute output on the body's normal range of physiological tolerance for water (reprinted with permission).[1]

only 3-4 liters (figure 1). A comparable individual on an average or high solute diet has a tolerance for water in the neighborhood of 15-20 liters. This means that an individual with normal regulatory function on a low or no solute intake will dilute his body fluids if he receives fluids in excess of

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Portions of this work have been done by Hans E. Jorgensen, M.D., Fulbright Fellow in Urological Research.

replacement and his physiological tolerance (approximately 2.5 liters per square meter surface area per 24 hours).

Before discussing how such a situation is changed when trauma is imposed, it might be useful to review briefly the present-day concept of kidney function which pertains to the relationship between the physiologic tolerance for water and urine-solute output.[2] In the normal individual an approximate 130 cc. of ultrafiltrate is formed every minute. From this is extracted a variable but large amount in the proximal portion of the nephron, the water supposedly being reabsorbed secondary to the active reabsorption of sodium. Apparently, no concentration nor dilution mechanism is to be found in the proximal tubule, so that the fluid left over from the proximal reabsorption is delivered to the distal tubule and collecting tubules where the change from isotonicity with serum takes place. The amount of nonreabsorbed solutes in the proximal tubule urine determines the amount of water delivered to the distal tubules for final work in regard to dilution or concentration. If sodium reabsorption is high and urea concentration low, as the case would be in an individual on a low solute intake, then the amount of water reabsorbed passively in the proximal tubule would be high, and the amount delivered to the distal tubule would be low. Concentration or dilution of such tubular urine would in a sense be below capacity for the system.

The concentration mechanism, that is, the reabsorption of osmotically-free water, is limited by a maximal capacity ($T^c_{m_{H2O}}$ approximately 5 cc/min.) which is usually not reached before urine volume exceeds 5-7 cc. per minute. If the load of iso-osmotic urine delivered to the distal tubule is less than $T^c_{m_{H2O}}$ water reabsorption is limited by the urine to plasma osmotic pressure ratio $\left(\frac{\text{Uosm}}{\text{Posm}}\right)$ which in man is about 4. Maximum utilization of the dilution and concentration mechanisms in the kidney can apparently be ascertained only when enough volume is delivered from the proximal segments of the nephron. Therefore, one way to increase the fluid volume reaching the distal tubule system would be to increase the amount of nonreabsorbed solutes in the proximal tubular urine. This, in essence, is what might be termed an osmotic diuresis and can be obtained by increasing the solute intake which, in a normal individual, could be in the form of an increased protein and/or salt intake. Under ordinary circumstances, 90 per cent of protein digested will be excreted in the form of urea. Consequently, an increase in the protein intake does constitute an increase in the solute intake leading to a higher solute excretion in the form of urea, whereby an osmotic diuresis ensues, resulting in an increase in the physiologic tolerance for water (figure 1).

The excretion of solutes on the operative day, according to Hayes and co-workers,[3] amounts to 205±60 milliosmols per square meter per 24 hours. This figure appears to be in close agreement with figures we have obtained in our studies. Such a low excretion of solutes, resulting from a low intake of solutes, decreases the tolerance for water. A much more severe restriction in the tolerance for water, however, is imposed by the almost obligatory antidiuresis which results from trauma.[3,4,5,6] The actual cause of this antidiuresis of trauma is not known, but it appears to be a result of release of posterior pituitary antidiuretic hormone. Whatever the cause, it is unrelated to serum osmolality and thus cannot be reversed by any attempt to lower the concentration of serum electrolytes. It is sufficient at this point to mention that antidiuresis following trauma in dogs and rats in our experience is at best very short-lasting (hours) and parenterally administered water will not lead to overhydration as the case is in man. It might also be added that in a few cases in the old age group we have observed patients who did not exhibit the usual antidiuretic response to surgery. These had very high pain thresholds, showed little or no apprehension, and in general had remarkable recoveries. Fluid therapy in such patients created no problem, but these are the exception rather than the rule.

The commonly observed post-traumatic antidiuresis usually means that there is no tolerance for water. Returning to figure 1, the water intake in such a patient should be

WEIGHT LOSS AND SOLUTE EXCRETION IN NORMAL CONTROLS DURING 24 HOURS OF INTRAVENOUS FLUIDS

Subject	Sex	Weight Loss grams	Solute Excretion milliosm.
P.J.	Male	2090	791
T.P.	Male	1449	661
J.H.	Male	1838	790
M.L.	Female	1000	428
A.Z.	Female	649	445
K.N.	Female	1146	489

Table I

identical with the minimal tolerable water intake. Any excess of water will lead to dilution of serum electrolytes and an expansion of body fluids. To evaluate this, it is essential that a baseline be established regarding weight and serum electrolyte changes in normal individuals on bed-rest, receiving parenteral fluids only. Evaluation of weight and serum electrolyte changes in post-operative patients must be compared with changes occurring in normal unoperated controls treated identically. In Table I are presented some baseline data from six normal individuals, three males and three females. They were on bed-rest and subjected to the routine procedures of no oral intake after midnight and from 9 a.m. the following morning they were given parenteral fluids consisting of dextrose in water. They were weighed before the intravenous fluids were started at 9 a.m., and again 24 hours later. The weight loss in grams and the amount of solutes excreted in the urine, expressed in milliosmols, are presented. All figures are corrected to 1.73 square meter surface area. The females appeared to lose somewhat less than the males. There were no changes in serum electrolytes.

A group of 18 surgical patients who received an approximate 2000 cc. of dextrose in water intravenously during the operative day (24 hours) showed a mean weight loss of 164±226 gm. The serum sodium in these rectly proportional to the excretion of solpatients on the day following surgery showed a decrease of 3.78±0.77 per cent, compared to the preoperative level. These fig-

Table II
PATIENTS RECEIVING 5 PER CENT DEXTROSE INFUSIONS WITH 4 PER CENT UREA.

Subject	Intake H_2O cc.	Urine per cent of intake	Urea excreted per cent of intake	Na balance meq.	Serum Na per cent change	Serum osmolarity (corrected per cent change)	BUN change mg. per cent	Weight change grams
			A. (Appr. 4000 cc. Intake)					
E.C.	4000	102.0	73.6	—126	—2.1	—1.4	+56.3	— 500
H.D.	4000	92.7	54.0	— 97	—2.1	—0.7	+34.7	—1100
C.H.	3845	95.3	69.7	— 29	—0.2	+1.5	+26.3	— 800
G.E.	4210	86.2	56.2	— 24	+2.5	+6.0	+65.5	— 900
R.L.	4004	100.0	72.4	—118	—1.6	—0.3	+41.7	—1350
H.F.	4000	81.5	46.5	+ 55	—0.4	+2.5	+68.9	—1650
A.L.	4000	93.2	71.5	—108	—1.8	—1.1	+48.7	—1750
C.S.	4100	72.1	36.7	— 9	+3.6	+1.0	+61.3	—1400
P.M.	4000	83.1	56.3	— 37	—1.7	+1.4	+60.5	—1150
H.F.	4000	93.8	54.5	+ 29	+4.0	+4.2	+35.3	—1900
Mean	4016	90.0	59.1	—46.4	+0.2	+1.3	+49.9	—1250
			B. (Appr. 3000 cc. Intake)					
C.W.	2750	87.3	57.3	+ 15	+2.4	—1.4	+48.5	— 450
H.H.	2590	93.6	52.4	— 51	—3.2	—1.4	+61.3	— 500
I.L.	3350	103.0	65.1	— 18	—0.3	+2.2	+39.2	—1400
H.W.	3300	87.1	66.2	— 6	—2.6	—6.1	+50.1	— 950
J.A.	3000	77.4	49.6	+ 13	+1.1	+2.1	+44.8	— 550
Mean	2998	89.7	58.1	—9.4	—0.5	—0.9	+48.8	— 770

ures, when compared with the normal controls, must indicate that all these patients have actually been overhydrated, their weight loss was less than that of the normal controls, and their serum sodium diluted.

To create an osmotic diuresis under such circumstances will not increase the water tolerance. However, it is well known that during antidiuresis the urine volume is dilute. We have shown that administration of 4 per cent urea intravenously given with dextrose and water will increase the volume output and that large amounts of intravenour fluids can be given in this fashion without any danger of overhydration.[6,7,8] Table 2 presents the data from two groups of patients subjected to suprapubic prostatectomies, who received 4 and 3 liters respectively of intravenous infusions containing 4 per cent urea. It will be noticed that the weight changes are rather close to those of the normal controls and there are practically no changes in either serum sodium or serum osmolarity.

In short, one might say that following trauma, the normal range of physiological tolerance for water does not exist. In fact, there is no range. The tolerance for water is nil, so only replacement can be given without running the risk of overhydration. In utilizing osmotic diuresis, one does not increase the tolerance for water but replaces water with a solution containing a solute that can be excreted by the kidney under the conditions of antidiuresis, meaning that the concentration of the solute given must be of approximately the same concentration as it is in the urine excreted. If one were to give higher concentrations than 4 per cent urea, dehydration would result. Dehydration could likewise result if large amounts of 4 per cent urea were given intravenously. However, this would rarely have practical significance since one would have to exceed 8 to 9 liters a day. ($T^{c}_{H\,O}$ does not become constant before urine volume exceeds 5 to 7 cc/min.)[2]

To summarize the question of tolerance for fluid on the day of surgery or the day of trauma, one might state that water cannot be tolerated in excess of replacement. However, water with a solute in suitable concentration, such as 4 per cent urea, can be given in any desired quantity with a maximum of an approximate 8 to 9 liters per 24 hours. We have thus not changed the lack of tolerance for water, but we still have tolerance for fluids such as 4 per cent urea, which can be utilized if a high urinary volume is desired. It might further be added that urea administration in amount and concentrations as used here does not change sodium excretion.[8]

Before discussing the situations where high urinary volume may be desirable or perhaps even imperative, it may be worthwhile to point out that use of osmotic diuresis in surgery has been made in this department, in a study on sodium retention following trauma.[9] It has been suggested that a lowered serum sodium level leads to retention of sodium, probably through an increased production of aldosterone. Since dilution of serum sodium occurs following most cases of trauma where severe restriction of water has not been emphasized, it was found useful to evaluate the sodium retention in patients given 4 per cent urea intravenously rather than dextrose in water, alone. In the previously mentioned 18 surgical patients who were given approximately 2000 cc. of dextrose in water, the serum sodium showed a drop amounting to 3.78 ± 0.77 per cent of the preoperative level. In 30 surgical patients, 4 per cent urea was given with dextrose in water, leading to weight losses more comparable to our control group of non-operated individuals (1164 ± 173 gm.) and no drop in serum sodium (-0.18 ± 0.62 per cent). This latter group, however, showed sodium retention, which must have been unrelated to serum sodium levels. This is inserted just to point out the value of the use of osmotic diuresis in surgery for the study of physiological phenomena associated with trauma.

Are there any situations in which the use of an osmotic diuresis in surgery is of real practical significance? The answer to this, I believe, is that there are situations in which a high urinary volume is to be desired and, at times perhaps, even essential. At other times, it probably is non-essential but practical.

It appears reasonable that, in any situation where nephrotoxic substances might be present, a dilution of such substances by promoting an osmotic diuresis could be beneficial. It has, for instance, been shown that the combination of hemoglobinemia, renal ischemia, and antidiuresis is dogs could result in renal damage, where none of the individual factors alone would result in such.[10] However, this damage was prevented when osmotic diuresis was established by the administration of mannitol.

In cases of gross hematuria, as seen for instance following urological surgery, an internal irrigation can be achieved by means of high urinary output feasible only following the trauma by an osmotic diuresis. We have thus given up to 5 liters of fluid with 4 per cent urea intravenously to patients following suprapubic prostatectomies during the first 24 hours of surgery. This is definitely a simplification in the management of these patients, since frequent bladder irrigations are no longer necessary and subsequently the dangers of infection with contaminated irrigation fluid are eliminated.

Crush injuries and thermal injuries constitute special problems since extensive replacement therapy is imperative but often difficult to evaluate accurately. According to Moore[11] a severe burn on the average, in an adult, when adequately treated usually results in a total weight gain approximating somewhere between 8 to 10 per cent of the patient's normal body weight. Hardy and co-workers[12] report weight gains in severely burned adults ranging from 5-12 kilograms. In Table III are presented weight changes in 8 burn cases given only enough saline and blood to maintain blood pressure and pulse rate. The amounts of saline given were in some cases only one-half to two-thirds of those recommended by the Brooke formula.[13] Further fluid therapy consisted of 4 per cent urea in dextrose and water in an amount sufficient to maintain a urine volume between 100-200 cc/hour. The serum sodium concentration was unchanged within the first 24-48 hour period following the burn, showing neither any increase nor any decrease ($+0.46 \pm 1.00$ per cent). The weight increases in these patients are far less than what is considered necessary in the light of formation of obligatory edema. We consequently feel that obligatory edema in burns is not as great as has hitherto been thought and that a closer approximation is reached when an osmotic diuresis is established with 4 per cent urea. It might be added that we feel that this approach to the treatment of burns appears to be a tremendous simplification and certainly completely eliminates the hazards of overhydration.

The use of osmotic diuresis in surgery for the purpose of dehydrating the patient, using concentrations in excess of those that can be handled by the kidney, can be applied in patients where previous overhydration has led to hyponatremia.[14] Dehydration of neuro-surgical patients with urea in concentrations from 5 to 30 per cent has been used with apparent benefit.[15,16]

Having discussed the usefulness and definite place in therapy for solute diuresis, whether it be urea, mannitol, sucrose or even low molecular weight Dextran, one must add

INITIAL WEIGHT GAINS IN BURN CASES GIVEN 4% UREA INTRAVENOUSLY

No.	Patient	Sex	Age	Extent of Burn %	36-48 Hours Weight Change %	Clinical Course
1.	D.R.	Male	3	50-60	+6.0	Died 10th Day
2.	H.S.	Male	37	20	+2.9	Recovered
3.	C.N.	Male	2	50-60	+9.3	Recovered
4.	C.B.	Male	44	40	+5.2	Recovered
5.	M.L.	Female	59	30	+4.6	Died 12th Day
6.	W.W.	Male	40	30	+5.8	Recovered
7.	A.C.	Male	41	60	+3.9	Died 9th Day
8.	T.B.	Female	5	45-50	+5.5	Recovered

Table III

that cases where such therapy is truly indicated actually constitute a minor portion of those seen by the general surgeon. The average surgical patient will have no particular benefit from an osmotic diuresis, but will usually do perfectly well if proper restriction of fluids is observed on the day of surgery as well as the following 2-3 days. Proper restrictions however, according to Hayes, et al.,[3] mean administration of only approximately 1700 cc. dextrose in water per 1.73 square meter surface area on the operative day in excess of possible replacement. We have, however, seen several patients receiving this amount exhibiting weight losses considerably less than those seen in our non-operated controls, and exhibiting dilution of their serum electrolytes. Quite frequently an intravenous drip is maintained, especially in patients where replacement of losses is necessary. If the drip is not maintained at a slow enough rate, overhydration under such circumstances frequently results. If the drip was maintained with a solution containing a solute such as urea in a concentration of 4 per cent, overhydration would be impossible provided the patient is not in shock. We therefore feel that, even in patients where the establishment of an osmotic diuresis is not of particular importance, the use of a solute in the intravenous fluid in a suitable concentration will insure the patient against overhydration and make the parenteral fluid therapy much safer.

In conclusion, it might be said that the use of the osmotic diuresis in surgery is a definite step forward. It has a usefulness, in certain cases, which can be life-saving. In other cases it might just be a safeguard against overhydration. The type of solute used is of little significance as long as it is a substance which will be excreted in the urine, since it otherwise would not act as an osmotic diuretic. We have found urea the most useful agent so far, since it is a physiologically-occurring substance with little or no toxic effect, which will distribute over total body water and thus can be given more safely to persons with impaired cardiovascular function. Mannitol and sucrose have the disadvantage of remaining in the extracellular space, thus by virtue of their osmotic effect causing an expansion of this space, which possibly could constitute a hazard in persons with borderline cardiac function. Urea has the disadvantage of being very heat-labile, requiring sterilization by filtration, and storage either in lyophilized form or under refrigeration. It further has to be given with dextrose or saline since urea in water alone can cause hemolysis. These latter disadvantages are, however, minor and cause no serious problems. We have for the past years used urea-containing solutions on many hundreds of patients and have never encountered any side effects nor difficulties of any sort.

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DISCUSSION

DOCTOR FREED, Assistant Professor of Medicine, University of Oklahoma School of Medicine: Doctor Schlegel, is there a commercial urea available for intravenous use?

DOCTOR SCHLEGEL: Urea is now available in lyophilized form from Abbott Laboratories.

DOCTOR COOKE: May I comment on urea?

DOCTOR FREED: By all means; go right ahead.

DOCTOR COOKE: There is one thing I would like to emphasize, and that is that not all patients can be assumed to have the capacity to concentrate their urine. I think this is particularly pertinent to the problem of the use of urea.

DOCTOR SCHLEGEL:: I would like to state that I did not emphasize that the use of 4 per cent urea was in people with normal kidneys. However, there is a very simple tool whereby this can be evaluated, and that is to do an osmotic pressure determination on the urine or just evaluate the response. There is one thing I think should be said in regard to giving urea. It is very difficult, if not impossible, to pull out any excess sodium that you have given. If you pull out too much water, however, such can be readily given back again.

The Problem of Water Intoxication in Pediatrics and Surgery

ROBERT E. COOKE

WATER INTOXICATION may be defined as the complex of neurologic symptoms resulting from a rapid decrease in the osmolarity of extracellular fluid. Since sodium chloride represents the bulk solute of extracellular fluid, water intoxication for all practical purposes results from a rapid fall in sodium concentration. Although much has been learned of the causes of hyponatremia, relatively little is known of the mechanisms whereby low sodium concentration leads to neurologic symptoms. Yannet and Darrow[1] demonstrated in cats that lowering of extracellular sodium concentration by peritoneal dialysis without change in total body water resulted in a shift of water from the extracellular to the intracellular compartment. The shift of water in liver and muscle was proportional to the fall in extracellular sodium concentration. By contrast no correlation existed between total water or intracellular water of brain and serum sodium concentration.[2] However, the potassium content of brain fell in proportion to the fall in sodium concentration. A decrease in serum sodium concentration of 1 mEq. per liter was accompanied by a fall in intracellular potassium concentration of approximately 1 mEq. per liter. No information was obtained as to the change in intracellular anions.

However, the neurologic symptoms associated with acute hyponatremia can not be explained on a basis of shifts in brain cell potassium. Utilizing the same experimental techniques Swinyard et al[3] noted a marked reduction of the threshold for seizures induced by electroshock or Metrazol® in hyponatremic rats. This effect could also be obtained without peritoneal dialysis by the administration of large water loads. Convulsions occurred spontaneously in animals subjected to both peritoneal dialysis and water loading. The increased sensitivity to epileptogenic stimuli was maximal 4 hours after paracentesis, could be reversed within 30 minutes by the administration of hywater was accompanied by a decrease in threshold and vice versa. This finding though considered statistically insignificant is consistent with Rowntree's[5] original observation that cerebrospinal fluid pressure was increased and cerebral edema present at post mortem examination. Nevertheless, in experimental studies the only significant correlation was between electroshock threshold and the concentration of sodium and chloride in serum. Expansion of the extracellular space with isotonic saline produced no change in threshold. Hypernatremia produced gradually by 96 hours of thirsting or ingestion of 5% saline did not elevate threshold. A small quantity of sodium chloride given as a 20% solution intravenously produced a 20% rise in threshold within 30 minutes. These experimental findings are consistent with clinical observations that severe hyponatremia may exist without neurologic disturbance if the changes in extracellular osmolarity occur gradually. Likewise signs and symptoms characteristic of water intoxication have been observed during recovery from hypernatremia even though the serum sodium concentration is still above normal values. Thus the appearance of symptoms seem more closely associated with rate of change of serum sodium concentration than absolute serum levels.

Mechanisms of Hyponatremia

A lowered concentration of sodium in the serum may result (1) from a decrease in the amount of sodium in the extracellular space (2) from an increase in the volume of extracellular water and (3) from a combination of the above two factors (Table 1). Hyponatremia does not usually develop rapidly enough in many of these conditions to be associated with actue neurologic symptons. However, if an additional large water load were given in any of these conditions symptoms might result.

I. Deficiency of Extracellular sodium
- A. Inadequate intake
  1. Low salt diet
  2. Parenteral therapy with glucose in water
- B. Excessive losses
  1. Gastrointestinal—vomiting, salivary, gastric, biliary, pancreatic drainage, diarrhea, resin therapy, enemas (especially megacolon)
  2. Genitourinary
     a. Intrinsic renal disease—chronic nephritis, acute tubular necrosis (recovery phase), nephrosis (diuresis).
     b. Extrinsic influences—mercurial diuretics, diamox®, hypoadrenalism.
     c. Arachnoid—ureterostomy.
  3. Skin
     a. Normal sweat
     b. Abnormal sweat — cystic fibrosis, adrenal insufficiency
  4. Parenteral—thoracentesis, paracentesis, burns
- C. Redistribution
  1. Potassium deficiency
  2. Trauma

II. Expansion of extracellular volume
- A. Excessive intake
  1. Parenteral fluid therapy — glucose in water
  2. Oral (with diminished output)
- B. Diminish output (usual intake)
  1. Renal
     a. Intrinsic—nephritis, nephrosis, tubular necrosis, prematurity.
     b. Extrinsic: (1) excess of antidiuretic hormone—acute central nervous system disease, pitressin therapy, surgery; (2) circulatory—heart failure, cardiovascular surgery, malnutrition.
  2. Skin—premature in high humidity

III. Expansion of extracellular volume and deficiency of extracellular sodium
- A. Excessive losses of sodium associated with retention of water, central nervous system disease, pulmonary disease, pitressin therapy, excessive water therapy.

Table I

pertonic saline and subsided spontaneously within 24 hours. Electrolyte analyses 4 hours after paracentesis revealed a much smaller change in intracellular potassium of brain than was noted at 24 hours.[4] Brain potassium also rose after administration of hypertonic saline. A slight increase in brain

Sodium Depletion

Water intoxication has been observed in infants who were maintained on a very low sodium chloride intake (Lonalac®) because of congenital heart disease. However, most infants may remain on such a regimen for months without developing hyponatremia. Of particular interest and importance in Pediatrics and Surgery a few years ago was the rather frequent occurrence of neurologic symptoms precipitated by the acute loss of sodium from the plasma into water introduced into the colon for therapy or diagnosis of megacolon.[6] Although gelatin in isotonic saline has been recommended for such enemas potassium deficiency may occur on this regimen. Such untoward effects are now avoided by the use of gelatin solutions made up in Darrow's K lacate solution. Salt losing nephritis is occasionally observed in children and acute hyponatremia may develop. The neurologic symptoms resulting from disturbances in osmolarity may be obscured by symptoms of uremia. In hypoadrenalism water intoxication is seen rarely and hyperpotassemia is usually a more important problem. Water intoxication has been observed in such patients after a water load which is not excreted well in the absence of glucocorticoids. Heat stress may also be a precipitating factor because of the high concentration of sodium in the sweat. Arachnoid-ureterostomy for hydrocephalus may be complicated postoperatively by water intoxication because of the loss of cerebrospinal fluid into the urine.[7] At least 35 mEq. (approximately 2.0 grams) of sodium chloride must be added to the usual diet to prevent sodium depletion in such patients. The excessive losses of salt in sweat of patients with cystic fibrosis of the pancreas are rarely associated with symptoms of water intoxication since there is no defect in water excretion and salt depletion develops slowly. Occasionally rapid removal of ascitic fluid followed by excessive thirst and ingestion of water precipitates acute neurologic symptoms.

Expansion of Extracellular Space

More commonly water intoxication results from excessive retention of water rather than loss of salt. A large positive balance

of water may result from excessive intake in the presence of normal or reduced excretion or diminished excretion in the presence of usual intake. Most of the cases observed by the author have been precipitated by excessive administration of glucose in water parenterally.[8,9] Nevertheless, symptoms would probably not have occurred if the usual water diuresis had resulted. Case 1 is an example of iatrogenic water intoxication precipitated by the postoperative administration of volumes of 5 per cent glucose in water possibly appropriate for an adult but not for a child of 4 years.

Case 1. T.C.

A 4 year old white boy weighing 13 Kg. was operated upon at the Johns Hopkins Hospital because of progressive exercise intolerance and increasing cyanosis. A right subclavian to pulmonary artery end to end anastomosis was performed for the correction of a Tetrology of Fallot. The surgical procedure was well tolerated. Only 180 ml. of Dextrose in glucose was given during the operation. After operation 800 ml. of 5% glucose in water was administered intravenously over a period of 12 hours and approximately 140 ml. of water was ingested. This intake was in excess of 360 ml. per 100 calories metabolized. Morphine .2 mg. was administered once during that period. Voiding was observed only once, 12 hours postoperatively. At this time a generalized tonic and clonic convulsion occurred, followed by unresponsiveness. Serum sodium concentration was found to be 117 mEq. per liter. Administration of 300 ml. of isotonic saline over 10 hours led to cessation of seizures and gradual clearing of the sensorium. No central nervous system residuals were observed.

A similar situation may occur when oral rather than parenteral fluids are given if a disturbance in the thirst mechanism as well as the excretory mechanism is present. Case 2 illustrates the dangers which may arise when ad libitum clear liquids are permitted in the early postoperative period or in disorders such as meningitis. In some of these patients significant lowering of extracellular osmolarity fails to inhibit thirst or anti-diuresis as would be expected from Verney's classic studies.[10] Likewise the finding of expanded body water rather than reduced volume makes it unlikely that the volume receptors suggested by Leaf et al[11] are activated and responsible for neurohypophyseal stimulation.

Case 2. L.J.*

A 6 year old white female weighing 16 Kg. was operated upon for ligation of patent ductus arteriosus. Two days before surgery serum analyses revealed normal electrolyte concentrations. Nembutal, morphine and scopolamine were given as preanesthetic medication. Nitrous oxide and cyclopropane-oxygen-ether were given over a 3½ hour period. During operation and for the next 18 hours 810 ml. of electrolyte free fluid was given. In addition she ingested in excess of 500 ml. of water and gingerale. This intake was in excess of 240 ml. per 100 calories metabolized. Morphine sulfate was given 4 hours after surgery. Voiding was observed only once in the 18 hours postoperatively. At this time a series of generalized tonic and clonic convulsions occurred. A few hours later the serum sodium concentration was 118 mEq. per liter. Although barbiturates had been without effect, 140 ml. of 3 per cent saline given over 3 hours led to immediate cessation of the seizures and rapid clearing of the sensorium. No residuals were observed.

Since water retention represents an imbalance between intake and output a brief analysis of the quantitative aspects of water excretion is required. The loss of water from the lungs and skin in the absence of sweating is dependent upon a number of variables such as environmental temperature and humidity, rate and depth of respiration, skin temperature etc. By simultaneous measurement of caloric production and insensible water loss it has been shown that evaporative water losses bear a more consistent relationship to caloric production than to surface area particularly in states of disease. Urinary water loss depends upon two factors: (1) the quantity and to a lesser extent the kind of solute presented for excretion; (2) the concentrating activities of the kidney at that moment. The quantity of solute excreted in the fasting state is determined by the rate of turnover of body tissues and bears a closer relationship to heat production than to surface area. For these reasons water loss and water requirements are expressed in terms of units of caloric turnover (per 100 calories metabolized) rather than square meters of body surface.

Insensible water loss amounts to about 45 ml. per 100 calories metabolized and the slight sweating that frequently occurs in a

*This Case as well as Case 3 have been reported previously.[8,9] Over 10 similar cases have been observed in the past 3 years, but in many quantitative data on intake and output are not available.

hospitalized patient in bed raises the usual evaporative losses to 55 to 60 ml. per 100 calories metabolized. Stool losses are normally only 5 to 10 ml. per 100 calories metabolized. The quantity of non-electrolyte solute in the urine during restricted food intake ranges from 10 to 15 mOs per 100 calories metabolized. Urinary concentration in infants and children ranges from just below 100 to slightly above 1000 mOs per liter.

It is possible then to calculate that if water without usually excretable solute (5% glucose in water) is administered in excess of 160 to 200 ml. per 100 calories metabolized excessive retention must occur even in the presence of excellent renal function. In Case 1 intake exceeded MAXIMAL excretory capacity by 200 ml. per 100 calories metabolized. Such a rate of administration in 24 hours would lead to a 13 per cent increase in body water and a fall in serum sodium concentration of 17 mEq. per liter. In the presence of some antidiuresis the fall in sodium concentration would be even more precipitous.

As indicated in Case 3 excessive retention of water may occur even when usual amounts of water and electrolytes are administered.

Case 3. D.B.

A 17 months old white boy was admitted because of H. influenzae type b. meningitis. Chloramphenicol, streptomycin, and Gantrisin® were administered. Water, sodium and potassium were given parenterally in amounts not in excess of the usual requirements of the average patient. Approximately 120 ml. of water and 3 mEq. each of sodium and potassium per 100 calories metabolized were administered. The intake amounted to 72 ml. per M^2 per hour. Twelve hours after admission restlessness followed by intermittent grand mal convulsions occurred. The serum sodium concentration was 121 mEq. per liter. Administration of 140 ml. of 3% saline and 100 ml. of whole blood led to prompt cessation of seizures and irritability. Recovery was progressive thereafter.

Water intoxication in surgery and pediatrics results more frequently from diminished renal excretion due to extrinsic influences acting on the kidney rather than to intrinsic renal disease. Although few measurements of anti-diuretic hormone (A.D.H.) levels in the urine or plasma have been made in patients with central nervous system infection or postoperatively, oliguria with relatively high urinary specific gravity has frequently been observed in the presence of adequate water intake. Factors such as massive stimulation of peripheral sensory receptors, circulatory readjustments, morphine or anaesthesia probably stimulate neurohypophyseal release of A.D.H.[12]

The quantitative aspects of the problem can best be appreciated by an hypothetical example. If A.D.H. release is sufficient to lead to a urine osmolarity of 1000 mOs per liter and if the usual renal solute load of 20 mOs per 100 calories metabolized is present (10 mOs non-electrolyte, 10 mOs electrolyte), an intake of water in excess of 80 ml. per 100 calories metabolized will lead to water retention in the absence of increased extrarenal losses. If the usual intake of 1200 ml. per 100 calories metabolized is given, the serum sodium concentration of a 10 kilogram child will be lowered approximately 15 mEq. per liter within two days.

In this discussion of renal solute no distinction has been made between electrolytes and non-electrolytes. Evidence is available that electrolytes obligate more water for excretion than non-electrolytes.[13] The quantitative aspects of this problem have not yet been worked out over a sufficiently wide range of concentrations to permit discussion.

Expansion of Extracellular Space and Sodium Loss

Retention of water leading to expansion of body water may increase sodium excretion markedly. Strauss et al[14] and Leaf et al[15] have demonstrated that sodium excretion may increase rapidly when extracellular volume is expanded even with fluid of low sodium concentration. Some increase in glomerular filtration rate may be responsible but it is also likely that aldosterone release is diminished by the increase in ex-

tracellular volume. Water intoxication has been observed when these phenomena occur rapidly as illustrated in Case 4.

Case 4. A.M.

An 8 year old white boy was admitted to the Grace New Haven Community Hospital because of growth arrest, polyuria and precocious puberty. Diagnostic studies and craniotomy demonstrated a pinealoma which could not be completely removed. Over a period of several months he had recurrent episodes of generalized tonic and clonic convulsions. Serum sodium concentrations at these times ranged from 105 to 120 mEq. per liter. The seizures responded promptly each time, except terminally, to the administration of small amounts of 3 per cent saline. Balance studies were attempted on several occasions. Although some losses were incurred because of lack of cooperation, a consistent pattern of metabolic response was observed as illustrated in Table 2. Administration of pitressin led to reduction in urine volume and water intake. A small positive balance of water with a gain in body weight of 1 to 2 per cent occurred. Serum sodium concentration which was uniformly high prior to pitressin therapy fell precipitously. Urinary sodium excretion which was low when urine volume and serum sodium level were high rose markedly and was quantitatively responsible for the sharp fall in serum sodium concentration and symptoms of water intoxication.

Abnormal regulation of thirst must be present in such cases of central nervous system disease. In addition the mechanism responsible for loss of sodium seems unduly sensitive to relatively slight expansion of body water. Repeated measurements of sodium excretion indicate that when urinary water loss is high and body weight low urinary sodium excretion is low and serum sodium concentration high. When water output is low (pitressin therapy) and body weight increases by 1 or 2 per cent urinary sodium excretion rises markedly and serum sodium concentration falls to very low levels. Symptoms of water intoxication appear sporadically and seem to bear a closer relationship to the rapidity of change in sodium concentration than the absolute level.

Treatment

The emergency treatment of water intoxication is similar regardless of the mechanisms leading to hyponatremia. The usual anticonvulsants are ineffective in treating the seizures of water intoxication. Hypertonic saline should be given intravenously in amounts* adequate to raise the sodium concentration about 5 mEq. per liter in two hours. Since hypopotassemia is associated in some patients with hyponatremia and since symptoms and signs of potassium deficiency (especially cardiac) may be increased by elevation of the sodium concentration potassium should be added when pretreatment serum potassium levels are normal or low. Some cases of hyponatremia are associated with severe hyperpotassemia[9] and caution must be exercised.

The subsequent therapy of the patient must be individualized. When volume is expanded limitation of water intake or use of alcohol to invoke a water diuresis is indicated. When sodium depletion is present salt intake must be increased.

Conclusion

Since severe brain damage may occasionally remain as a residual of water intoxica-

* $\frac{(Cd - Ca) \times fd \times B.W}{(Concentration\ desired - C.\ actual) \times distribution\ factor \times body\ weight} = mEq.\ of\ Na$

$5 \times 0.6 \times B\ Wt \times 2 = ml.\ of\ 3\%\ NaCl.$

Sample Time	Metabolic Data Case 4 A.M.					
	Average Intake per day		Average Output per day		Body Weight Kg	Serum Na mEq/l	Remarks
	H_2O g	Na mEq	H_2O g	Na mEq			
11/23	600	35	300	29	15.5	146	Pitressin 0.4 ml b.i.d.
11/25	800	35	640	120	15.7	141	"
11/29	700	35	400	136	15.9	120	Convulsions
12/16	4500	35	4900	15	15.5	132	

Table 2

tion, prevention of this disorder is essential. As indicated in earlier discussions prevention depends upon the supply of adequate electrolyte to meet abnormal losses and limitation of water intake so that excessive retention does not occur. Total metabolism of the body varies considerably for a given body weight or surface area in pathological states such as fever, hypothermia, endocrinopathies. Since the turnover of body water and electrolyte is related to total metabolism, maintenance requirements for these materials, just as for protein and calories, should be calculated in terms of this parameter rather than surface area or body weight. Even with such a system of calculating therapy severe derangements in body composition will occur unless disturbances in the usual homeostatic mechanisms are suspected in specific clinical states.

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Central Nervous System Factors in Water and Electrolyte Metabolism

JOHN H. BLAND

PRIMARY METABOLIC DISORDERS may exert strong influences on central nervous system function; and certain structures within the central nervous system may produce profound metabolic effects. Disease and trauma of the brain has been associated with abnormal water and sodium metabolism; potassium depletion; azotemia and acidosis.

It is generally accepted that oliguria and retention of sodium accompany nearly all serious illness or trauma, related or unrelated to the central nervous system. There is much evidence to suggest that the stimuli producing salt and water retention are mediated via the central nervous system to the hypothalamus; then a neuro-endocrine stimulus reaches endocrine glands which secrete hormones regulating renal excretion of water and sodium.

This discussion will be confined to two areas:

(A) The central nervous system factors, known and postulated, normally regulating water and sodium metabolism.

(B) Hyperosmolar syndrome of central nervous system disease and its relationship to the normal reaction to dehydration.

PART I

Relationship of Hypothalamus, Posterior Pituitary Gland, Anti-Diuretic Hormone and Water

Antidiuretic hormone of the posterior pituitary gland is the principal agent regulating water excretion. Its action is on the distal tubule of the kidney. It promotes reabsorption of the solute free water.* Through the sustained efforts of du Vigneaud and his associates[1,2] the two hormones, Pitressin (ADH) and Pitocin (Oxy-

*Urine volume may be divided into two parts, osmolar water and solute free water. Osmolar water is that part necessary to contain urine solutes in a solution isoosmotic with plasma. solute free water is the remaining urine volume. Solute free water is reabsorbed from the distal tubule with ADH action: it is retained in the excreted urine during water diuresis (ADH suppression).

tocin) were purified and recently synthesized. They are both polypeptides containing eight amino acids per molecule. Six amino acids in the two hormones are identical; two are different.

It is unsettled whether these hormones are separately secreted or represent fragments of a single large protein molecule originally possessing activities of both. The fact that antidiuretic properties have been noted in plasma proteins,[4] might be construed as evidence for secretion of large molecular proteins from the pituitary gland. Small polypeptides, however, may be released and subsequently bound to plasma proteins as transport mechanism.

The current concept regarding posterior pituitary hormones is that they are secreted by the neurones of the supraoptic and paraventricular nuclei; then transported distally along the axones to the posterior lobe for storage in nerve endings close to blood vessels in this area.[4] The posterior lobe probably serves as a reservoir of manufactured hormone available for instant release into the systemic circulation as needs arise. This concept was a giant step in endocrinology and directs attention to the hypothalamus as the structure governing water metabolism through the pituitary body.

Verney[3] clearly established regulation of water excretion by an osmoreceptor located in the distribution of the internal carotid artery. The osmoreceptor is sensitive to osmotic pressure changes in the plasma. It modifies rate of ADH release in accordance with state of hydration. With increase in osmotic pressure, hyperosmolarity, and consequent movement of water from cells (cellular dehydration) antidiuretic hormone is released. With fall in osmotic pressure (hypoosmolarity) ADH release is suppressed.

Precise location of the osmoreceptors is not established. There is some evidence that they exist in the supraoptic nuclei in an intracellular position.[29] Dingman,[4] suggests that the osmoreceptor site may be in the posterior lobe. This is based on observations in patients who have had hypophysectomy as therapy of carcinoma. In such patients osmoreceptor control of available ADH was apparently lost or much diminished. The osmoreceptor response to hypertonic saline is absent more frequently than present—or else not as prompt and sustained. Osmoreceptor activity may depend on the saving of a part of the posterior lobe or pituritary stalk.

Another somewhat obscure pathway to the posterior pituitary involves a "volume receptor." A volume receptor promoting both sodium and water retention has been located somewhere in the head portion of the vascular tree. It is stimulated by a decreased circulating or effective blood volume; it is suppressed by expansion of blood volume. Prompt secretion of ADH in response to hemorrhage has been observed.[30] This suggests an important role of the posterior pituitary and ADH in regulating volume as well as concentration. Retention of water by volume receptor stimulation by hemorrhage may be sometimes inhibited by previous alcohol administration.[31] This maneuver, however, does not block the retention of sodium. This suggests, at least, that sodium retention may not be mediated directly through ADH via volume receptor stimulus. Presumably a decline in blood volume constitutes a stimulus to aldosterone release, resulting in marked renal sodium retention; an expansion of blood volume suppresses aldosterone release with consequent sodium diuresis.

Oxytocin is also released in large amounts on posterior pituitary stimulation. It has definite effects on sodium excretion.[5] Though the posterior pituitary is not presently implicated strongly in sodium retention this may prove to be the case as more knowledge accumulates regarding the relationships between the two hormones.

Some anti-hypertensive drugs produce water retention, presumably because of their influence on renal circulation or autonomic outflow. However, some of these drugs have potent effects on the mid-brain. They may thus facilitate water retention by stimulating ADH release. This has recently been claimed for hexamethonium.[6]

ADH release is familiarly produced by emotional, physical and physiologic stimuli

and by numerous drugs, e.g., morphine, acetylcholine, nicotine and barbiturates. Dingman[4] et al. have shown that adrenocortical hormones may influence ADH release in man.

Many seemingly divergent factors influence ADH release through hypothalamic mechanisms. It would also seem that stimuli may arise through a variety of avenues, e.g., osmoreceptor, volume receptor, possible alteration of vascular supply of hypothalamic nuclei, direct stimulation of nuclei by drugs or hormones. Nervous impulses may affect nuclei directly; volume impulses influence hypothalamic and posterior pituitary circulation; osmoreceptor stimuli may possibly be confined to the posterior lobe but other stimuli are mediated through hypothalamic nuclei to the posterior pituitary gland.

Dingman[4] formulates that the hypothalamus may be in the position of monitoring stimuli, permitting those of greater intensity or biologic necessity to dominate posterior pituitary activity.

Three major mechanisms of promoting ADH release in order of priority seem to be:[4]

(1) Neurogenic stimulation

(2) Osmoregulation

(3) Volume regulation.

All three may act in combination. It seems probable that inhibitory impulses suppressing ADH release are effective only when stimuli promoting release are quiescent or absent. Volume contraction or neurogenic stimuli promote ADH release in the presence of hypotonicity (a suppressant stimulus) suggesting that they are in higher priority or a stronger stimulus. Nervous impulses take precedence over inhibitory effect of an expanded volume. Hypertonic contraction of blood volume is a much more potent antidiuretic stimulus than is hypertonicity alone; still hypertonic expansion of blood volume if acutely induced evokes ADH discharge. This suggests precedence for osmotic stimulating impulses over inhibitory increased volume impulses. This order of priority is as expected if the primary function of the posterior pituitary and its control of water metabolism is:

(1) Initially to protect and preserve tonicity.

(2) More fundamentally to preserve volume.[4]

Relationship of the Anterior Pituitary Gland, Adrenal Cortex and Sodium Metabolism

The adrenal cortex is the major endocrine structure regulating body content of sodium. Adrenal cortical steroid hormones influencing electrolyte metabolism in man are hydrocortisone, corticosterone (Compound B) and 18 aldocorticosterone (aldosterone). Hydrocortisone and corticosterone are the principal secretory products and have been estimated to be secreted into a ratio of about 12 to 1 normally.[5]

Human adrenal gland has been estimated to secrete 17 to 29 mgs. of hydrocortisone and 2-3 mgs. of corticosterone/day. Beef adrenal gland contains only 45-95 micrograms per kilogram of aldosterone. Only traces are found in normal venous blood or urine. The present exciting investigations regarding aldosterone and its mechanism of secretion may have overshadowed importance of relationships of this hormone with hydrocortisone and corticosterone in understanding sodium metabolism.

Hydrocortisone[32] and corticosterone[33] secretion have been shown to follow a diurnal rhythm, highest in the early morning, falling sharply in the late afternoon and reaching its lowest level during sleep. A similar diurnal rhythm in excretion of aldosterone has not been confirmed.

A diurnal pattern for sodium excretion, mainly resulting from alterations in renal tubular reabsorption follows just behind the adrenal cortical rhythm of hormone secretion by only a few hours;[34] its peak occurs in the forenoon or at noon; its lowest level is during the night; this suggests an interrelationship between rhythmic adrenal secretion and sodium excretion.

Though one thinks of hydrocortisone, corticosterone, meticorten and cortisone as being sodium retaining steroids Ingle and

Thorn[9] and Sweat[10] have shown that very small doses of these steroids promotes loss of soduim by a decreased renal tubular reabsorption. More recently this has been studied with intravenously administered steroids in small doses. A sodium diuresis in normal subjects has been demonstrated. Retention of sodium was associated only with administration of large doses.[2] Administration of intravenous hydrocortisone in a dose of 1 mg. per hour to a patient with Addison's disease over a period of 24 hours increased renal excretion of sodium during the day and blocked sodium retaining effect of a small dose of DCA at night.[2]

These studies with physiologic doses of adrenal steroid hormone suggest that diurnal variation of sodium excretion in normal subjects is related to a rhythmic secretion of hydrocortisone influencing renal tubular reabsorption of sodium. The decline at night in sodium excretion may be related to unopposed effects of potent sodium retaining steroid as well as decrease in secretion of hydrocortisone.

Extracellular water volume influences tubular reabsorption of sodium. Contraction of ECW leads to marked sodium retention presumably by volume receptor stimulation. Expansion of ECW results in sodium diuresis, presumably by volume receptor suppression. Adrenal steroids frequently produce expansion of ECW at expense of ICW volume. Such expansion of ECW could promote sodium diuresis by suppression of volume receptor.

It is possible that a diurnal rhythm in ECW volume parallels secretion of hydrocortisone leading to a sodium diuresis during the day and retention at night. Methods of measuring extracellular water volume are not sufficiently accurate to study this effectively.

Aldosterone has been extensively studied recently. It has 20 to 35 times more sodium retaining power than DCA. Increased amounts of this steroid in blood and urine in edematous states has sparked an intensive investigation of its possible role in edema.

Aldosterone secretion is increased by sodium depletion; by administration of potassium; increased production is stimulated by contracted ECW volume and suppressed by an expanded ECW volume.[35] Factors related to volume seem to have primary importane in the regulation of aldosterone secretion. There is still controversy regarding the relation of the anterior pituitary to the secretion of aldosterone. Secretion continues despite anterior pituitary insufficiency. It increases when salt is restricted in such patients. ACTH does cause increased secretion of aldosterone to some degree in normal man. Recently Farrell[11] and colleagues demonstrated a diencephalic substance regulating this secretion. They showed that highly purified corticotrophins enhance it but commercial ACTH preparations do not. Beck[12] and associates showed aldosterone secretion to be increased in man on growth hormone administration (monkey and human growth hormone).

These studies suggest that trophic hormones of hypothalamic or pituitary origin may regulate production of aldosterone. Its continued secretion apparently is assured in the absence of the pituitary gland by such local factors as volume or concentration of ECW.

Guillemin[36] has described studies on neurohypophyseal polypeptides resembling pitressin. It is postulated that these compounds may stimulate release of ACTH from the anterior lobe. It is suggested that areas of the hypothalamus may control not only ADH secretion but also secretion of electrolyte regulating adrenocortical steroids. Neurohormones of the hypothalamus might govern aldosterone secretion, suggesting a possible role of the hypothalamus in sodium metabolism. Definitive studies of precise hypothalamic mechanisms are too few to permit other than speculation.

PART II

Hyperosmolar Syndrome of Central Nervous System Disease and Trauma and Its Relation to Normal Reaction to Dehydration

This discussion will be prefaced by a brief review of the expected, predictable changes

in the central nervous system and circulation initiated by ordinary, moderate to severe water depletion, uncomplicated by central nervous system disease. When simple dehydration occurs in the patient who also happens to have central nervous system disease or injury, on tends to construct a cause and effect relationship between cerebral lesions and the clinical and metabolic picture. When one is dealing with the same type of metabolic deviations, in the absence of any central nervous system dysfunction, he is unlikely to consider cerebral mechanisms as cause.

Body water depletion occurs most commonly in patients who for any reason do not receive or retain an adequate daily supply of water, e.g., comatose or semi responsive patients, children, patients with upper gastrointestinal obstruction or mentally disturbed patients who refuse fluids. The initial event in primary dehydration is rise in extracellular sodium concentration. There is immediate movement of water from cells disturbing the water deficit between cells and ECW. Reapportionment of the water deficit is mediated by the following mechanisms:

(1) Movement of water from cells in response to increased osmolarity of ECW.

(2) Release of cell potassium to ECW with consequent movement of cell water.

(3) Decrease in osmotically active cell cations—with release of further cell water to support ECW. This is simple dehydration.

If dehydration continues the adjustments are not adequate to prevent onset of the physiologic effect of hypertonicity and dehydration. Little change in the circulation occurs early. With moderate to severe dehydration, cardiac output decreases, circulation time is prolonged and there are slight falls in blood pressure. Circulatory collapse is rare because ECW and plasma volumes are so well maintained at the expense of cell water.

Though circulatory changes are minor, central nervous system manifestations occur with considerable regularity in patients with hypertonicity and dehydration. This is expected since central nervous system cells are involved in the general dehydration and hypertonicity and represent the first tissues to show functional change when metabolic necessities such as oxygen and glucose are not available in required amounts. The consequences of advanced cell dehydration was examined by Winkler et al.[17] in animals infused with hypertonic saline to death; death occurs when the pulse is full and bounding and the circulation is well maintained. The same events probably occur in castaways who drink and retain sea water or in patients deprived of water. Broch[18] in 1948 reported hypertonicity and hypernatremia in hospitalized patients following cerebral vascular accident. They received less than adequate amounts of water. Respiratory failure in this group of patients would be assumed to represent a consequence of the cerebral lesion. It might well be related to progressive dehydration. Since Broch's report there have been a number of papers on the hyperosmolar syndrome of central nervous system disease and trauma.[19, 20, 21, 22, 23, 24, 25, 26, 27] Examination of available case reports discloses no case whose clinical and metabolic features are not explicable in terms of the normal reaction to dehydration and to the physiologic consequences of water deficit. This does not, of course, rule out the possibility of a cause intrinsic to the central nervous system.

There can be said to be four types of hyperosmolarity in patients with central nervous system disease.

(1) Simple dehydration in which water requirements are not unusual but fail to be met adequately because of failure of the patient's attendants to evaluate losses and supply adequate amounts.

(2) Hyperosmolarity secondary to unusual demands for water because of either failure of thirst mechanism or failure of hypothalamic-posterior pituitary system to

control renal tubular reabsorption of water, diabetes insipidus. If the patient is unconscious and cannot exhibit thirst polyuria exceeds polysipsia.

(3) Dehydration secondary to unusual demands for fluids because of an especially high solute load presented to the kidney. This occurs in the unconscious patient fed by intragastric tube a diet containing relatively greater amounts of protein without a quantity of water sufficient to excrete nitrogen from the diet. This dehydration will be accelerated if the patient is in negative nitrogen balance or if he has some degree of renal insufficiency preventing excretion of urine of maximal specific gravity. Normal renal response to restriction of fluid intake with an increased nitrogen load is to excrete a urine maximally concentrated, containing a large quantity of urea and very little sodium and chloride. A fairly large volume of high specific gravity urine is excreted, serum shows increasing hypernatremia. As this dehydration progresses, there is, of course, contraction of extracellular volume, diminution in renal blood flow, azotemia and ultimate renal acidosis. The intensity of hyperosmolarity is a function of the discrepancy between water intake, solute load and the renal function capacity. If there is impaired renal function this syndrome may develop rapidly, associated with marked azotemia.

It is apparent that the three types of hyperosmolarity thus noted may occur in differing magnitude in the same patient—all may contribute to a given clinical and metabolic picture.

(4) The last type of hyperosmolarity is sometimes thought to be due directly to cerebral lesions. The supposition that specific cerebral lesions may be responsible for hypernatremia has some experimental support. Stevenson et al.[13] ablated the thirst mechanism and produced hypernatremia by experimental production of bilateral lesions in the ventromedian nuclei of the hypothalamus in rats. Lewy and Gassman[14] noted hyperchloremia in cats after unilateral stimulation of the paroptic nuclei of the hypothalamus.

A direct neural connection between the hypothalamus and the kidney has been postulated.[15,25] It was suggested that a neural connection modifies proximal tubular reabsorption of sodium and chloride. The syndrome of "cerebral salt wasting" has been explained by assuming that the defect is caused by diminished proximal tubular reabsorption of sodium and chloride because of interruption of a neural pathway between hypothalamus and kidney. As a consequence sodium, chloride and water are delivered to the distal tubules in excess. This exceeds the distal tubular reabsorptive ceiling and a continual osmotic diuresis proceeds. Ultimate ECW hypoosmolarity occurs. A similar direct neural connection has been suggested to explain the avid reabsorption of sodium in hyperosmolarity associated with cerebral disease.

If a metabolic or neural mechanism leading to hyperosmolarity due directly to brain lesions occurs or exists it is certainly obscure. It is difficult to evaluate case reports of cerebral damage and hypernatremia; balance data may be lacking. Such patients commonly have intragastric tubes placed in the stomach and may be receiving a relative excess of protein. Azotemia has been commonly reported but it is difficult to rule out renal disease. Potassium depletion has also been reported though less frequently.[27]

Many features of the hyperosmolar syndrome of cerebral damage are entirely consistent with severe dehydration. Hypernatremia, hyponatruria, hypokalemia, hyperkaluria, azotemia and acidosis are all characteristic of advanced dehydration, (severe water depletion). In varying degree this is the biochemical picture in hyperosmolarity of cerebral damage. It has been noted that clinical signs of dehydration are not apparent in these cases. However, since water loss is mainly from cells physical signs of dehydration would appear late, after marked extracellular contraction. Central nervous system signs precede signs of dehydration and circulatory failure. This may be masked by brain lesions.

Comatose neurological or neurosurgical patients are very apt to develop severe wat-

er deficit with much less of a sodium deficit. They often do not experience or express thirst. Fever, sweating and hyperpnea are common in such patients. All these accelerate water loss. The magnitude of sweat water loss is easily underestimated. Hyperventilation water loss is difficult to quantitate. Involvement of the medullary respiratory center may cause hyperpnea. Such insidious loss goes unnoticed. Extrarenal water loss may develop rapidly or gradually. Fever may be due to involvement of the hypothalamic thermoregulatory center. Excess sweating may likewise be caused by a lesion involving the thermoregulatory center. Thus, in a sense the metabolic changes may be indirectly caused by central nervous system lesions.

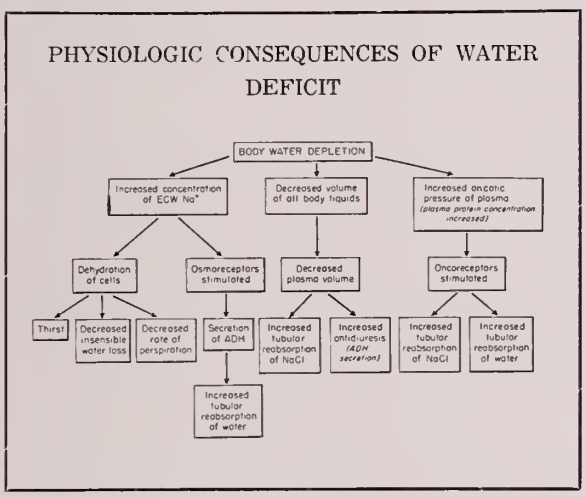


Figure 1

Figure 1 outlines the physiologic consequences of water deficit as outlined and documented in a series of responses formulated in the classic review article by Welt[16] and associates. The first and most important response to water deficit is a rise in sodium concentration of ECW. Immediate transfer of water from cells occurs; ICW compartment repairs the water deficit in ECW. The consequent cellular dehydration constitutes a stimulus to decrease in sweat rate and insensible perspiration. It also initiates thirst sensation.

The hyperosmolarity of ECW results in rise in cellular osmolarity. This stimulates hypothalamic osmoreceptors promoting increased release of antidiuretic hormone from the posterior pituitary increasing renal tubular water reabsorption.

Decline in volume of total body water includes plasma volume contraction. This stimulus promotes increased renal tubular reabsorption of sodium and chloride. It is postulated that this is by the mechanism of a volume receptor, stimulating aldosterone release. This is the most recently documented consequence of water deficit. Striking sodium retention, augmented potassium excretion and ultimate metabolic alkalosis occurs if other forces are not present. Years ago Doctor Peters called this the "reaction to dehydration." Contracted plasma volume is also a further strong stimulus to antidiuresis via the posterior pituitary.

Oncotic pressure of the plasma increases. This likewise is a stimulus to increased tubular reabsorption of both salt and water. Depletion may be augmented by tube feeding of a relatively excessive protein intake. This is especially true following trauma, during the catabolic phase in which the patient is unable to utilize nitrogen. Nitrogen freed in the catabolic phase of the response to trauma plus the urea solute load from the diet places added demands on endogenous sources of water. If dehydration proceeds and plasma volume continues to contract, renal blood flow decreases, oliguria occurs and ultimately, and quite late, peripheral vascular collapse follows.

Thus, a convincing picture can be formulated that the clinical and biochemical features of the hyperosmolar syndrome of cerebral damage may, at least sometimes, constitute an excessive but normal response to extreme water depletion.

It seems that one need not always invoke a cerebral lesion as directly causative of the hyperosmolar syndrome. Severe dehydration can occur readily in the unconscious patient via fever, hyperventilation, sweating, inappropriate amounts of protein in tube feeding and inadequate estimates of water needs. With advanced water depletion it is possible that secondary renal tubular damage may occur due to contracted plasma volume and inadequate renal blood flow. The

hypernatremia, hyponatruria, azotemia, potassium depletion and even acidosis can be explained as a physiologic response to severe water depletion.

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DISCUSSION

DOCTOR FREED: Doctor Bland, can you please outline briefly the best method for treatment of the hyperosmolar syndrome, and does this occur in cerebral vascular accident cases very frequently?

DOCTOR BLAND: The treatment of the hyperosmolar syndrome may vary somewhat, depending on the etiology of its occurrence and the magnitude and duration of extracellular hyperosmolarity, as measured by sodium concentration of serum.

The two main aims in treatment are (a) to provide water as such, (b) to administer as little solute as possible until the hyperosmolarity is well along toward correction.

Water may be given as water by mouth or as a 5 per cent glucose in water by vein. Progress may be gauged by examining serum sodium concentration, urine specific gravity, urine sodium concentration, which is low or absent in advanced hyperosmolar syndromes. Clinical signs observed at the bedside serve as an excellent guide.

A cautionary note should be added. Patients with advanced hyperosmolar syndrome of any cause are usually potassium depleted and in acidosis. Thus the addition of potassium salts as well as sodium bicarbonate may well be indicated.

In regard to frequency of occurrence, I believe it to occur rather more commonly than it is recognized. It occurs frequently in a mild form in many tube-fed patients.

Many such patients, e.g. cerebrovascular accident and head injury, are critically ill. If they are comatose and tube-fed, hyperosmolarity becomes a greater risk. The clinicians tendency wold be to ascribe death in such patients to the cerebral lesion rather than any metabolic cause. I have wondered if some patients in this category dying of respiratory failure might not be dying of hyperosmolarity, potassium depletion and acidosis, respiratory failure being a consequence.

DOCTOR FREED: In the intravenous therapy of an individual with brain injury, are there any danfers in the use of 50 per cent glucose solution, 5 per cent glucose solution, and 10 per cent glucose solution?

DOCTOR BLAND: Yes, I think there is potential danger in using any of those three solutions in a patient with head injury. It has been shown that changes in osmolarity of extracellular fluid, e.g. plasma, is followed promptly by change in hydrostatic pressure of cerebrospinal fluid.[1] With an increase in plasma solute concentration, there is a decrease in cerebrospinal fluid pressure; with decrease in concentration of plasma solutes, there is an increase in cerebrospinal fluid pressure.

In the case of 50 per cent glucose given intravenously, there is an initial sharp rise in glucose concentration and a fall in cerebrospinal fluid pressure due to movement of water out of the cerebrospinal fluid space into the plasma. However, as the glucose is rapidly metabolized and insulin secretion rises, the blood sugar concentration falls below the concentration of sugar in the cerebrospinal fluid; this event favors transfer of water from the plasma to the cerebrospinal fluid with rise in cerebrospinal fluid pressure. This observation has led to the abandonment of use of 50 per cent glucose in such patients.

Much the same application might be made in the case of either 5 or 10 per cent glucose in water. It has been shown that administration of either in amounts of a liter or more results in an increase in cerebrospinal fluid pressure.[2]

DOCTOR FREED: Would you explain the mechanisms of that?

DOCTOR BLAND: The cerebrospinal fluid pressure is elevated after 5 per cent glucose infusion because water moves from the plasma to the cerebrospinal fluid space; the glucose is very rapidly metabolized or converted to glycogen. Hence, a large volume of water is metabolically divested of its solute (glucose) which was responsible for its original tonicity. The added water volume to the plasma renders it hypotonic; with this decrease in osmotic pressure of extracellular water, an osmotic gradient results; this favors transfer of water to cerebrospinal fluid, resulting in a rise in intracranial pressure.

I suggest that the best solution to give intravenously to the head injury patient is 2.5 per cent glucose in 0.42 per cent saline (half hypotonic). This solution exerts little or no influence on cerebrospinal fluid pressure.

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Stress: Its Effect Upon Fluid and Electrolyte Metabolism

WILLIAM W. SCHOTTSTAEDT
ROBERT BARNES

THAT STRESSFUL SITUATIONS and emotional reactions are associated with variations in urinary volume and urinary composition is not a new observation though it has been the subject of investigation only in recent years. In 1924 MacKeith[1] reported that tension preparatory to running a race was associated with inhibition of urinary output quite similar to that seen during running itself. In the following year Mohr[2] published observations demonstrating that a marked diuresis occurred during periods of anxiety. Mohr also suggested the possibility of increasing the diuretic effect of drugs by making use of those emotional states which were associated with diuresis. These observations were extended in 1938 by Rydin and Verney[3] who concluded from their experiments that the anti-diuresis of mild exercise was a function of the stress experienced when exercise was begun. Eight years later O'Conner and Verney[4] published the results of experiments which demonstrated that sympathetic stimulation and secretion of anti-diuretic hormone were two factors in producing this anti-diuresis. Brod and Sirota[5] demonstrated a decreased renal blood flow and decreased glomerular filtration rate in rabbits during the emotional disturbance produced by noxious stimulation. And Blake[6] reported in 1951 a decrease in sodium excretion in association with exercise and "emotion" in dogs. In this same year Hinkle, Edwards and Wolf[7] reported a diuresis in normal and diabetic individuals occurring in association with feelings of anxiety, confirming the report of Mohr.

The studies to be summarized here began as an attempt to arrive at a more precise delineation of the fluctuations in renal excretion which accompany stressful situations.[8] These investigations began with a study of five healthy subjects who collected urine specimens four times a day while going about their usual daily activities. Collections were continued for 18 to 42 consecutive days. No restrictions were imposed

upon the subjects but the major pertinent variables were carefully observed. Thus the individuals recorded food and fluid intake (both the amount and time of taking them), position, activities, events of the day, and their emotional responses to these activities and events.

It was striking to observe how minor was the influence of ordinary variations in intake and physical activity upon renal excretion during short term observations under these conditions. The data for the study as a whole clearly showed that the general level of intake determined the general level of output when periods of two to six weeks were being considered; but this was not true for periods of five or six hours.

Table 1 illustrates this fact for urine volume. With a fluid intake of 300 cc. or less in the morning, morning urine volumes ranged from 75 to 663 cc.; with a fluid intake of 500 to a 1000 cc. in the morning, urine volumes ranged from 108 to 642 cc. Thus, there was a marked similarity in the ranges of output despite very different levels of intake.

MORNING FLUID INTAKE AND OUTPUT IN FOUR SUBJECTS

N	Intake	5-hour urine volume
25	0- 299 cc.	75-663 cc.
43	300- 499	78-600
34	500-1000	108-642

Table 1

Table 2 gives illustrative data for sodium excretion. It demonstrates that similar excretion rates were observed at low and high levels of sodium intake. And Table 3 demonstrates a similar phenomenon for potassium.

Since we know that sodium can exert a retaining effect upon water, one would expect that the answer to the discrepancies noted between intake and output might be explained by combining the data. In Table 4 we have taken the data from a subject studied for 42 days. Morning specimens only have been used to avoid any influence

SODIUM INTAKE AND OUTPUT IN SUBJECT No. 1

N	Intake	5-hour output
86	0- 50 mEq	5-63 mEq
46*	50-142	9-68

*One specimen omitted

Table 2

MORNING POTASSIUM INTAKE AND OUTPUT IN SUBJECT No. 1

N	Intake	5-hour output
21	0- 5 mEq	5-27 mEq
25	6-17	7-25

Table 3

of diurnal variation. There were 29 mornings with a fluid intake of 300 cc., but with considerable variation in sodium intake. The range of excretion rates is essentially similar for the observation periods with low sodium intake as for those with high sodium intake. These data are shown to document

MORNING SODIUM AND FLUID INTAKE AND OUTPUT IN SUBJECT No. 1

N	Fluid intake	Sodium intake	5-hour urine volume
15	300 cc	0-15 mEq	81-534 cc
14	300	16-45	105-489

Table 4

the statement that the immediately preceding intake has only a minor influence on output under the conditions of ordinary daily living.

Nor can one show a clear association of output with activity or rest. In Table 5 are given data relating to unusual daytime exertion or to daytime recumbency for periods of two hours or more.

Again, one notes a very similar range of excretion rates under either set of circumstances, some rates being very low and others quite high. Levels of intake are not shown in this table but combining intake

VARIATIONS IN ACTIVITY AND RENAL EXCRETION IN SUBJECT No. 1

Condition	N	(——5-hour output——) H_2O cc	Na mEq	K mEq
Quiet routine	47	153-339	19-42	10-23
Unusual physical exertion	8	96-471	4-42	14-26
Prolonged daytime rest period	10	90-519	11-40	14-21

Table 5

data with excretion rates does not affect the interpretation.

The marked variations just discussed do show a rather striking correlation with the emotional state of the individual, however, and this without regard for level of intake or amount of activity. Periods of tranquility have been chosen as a baseline for comparison with other emotional states. These have been defined as periods during which the individual was going about routine and familiar activities without awareness of any disturbing influences. Figure 1 shows com-

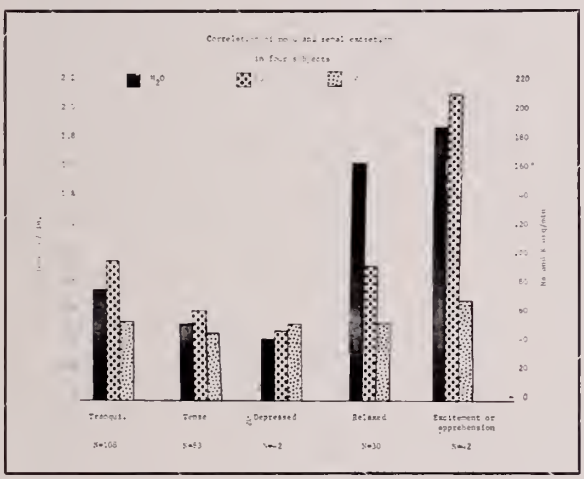


Figure 1

bined data for four of the five subjects. The fifth subject could not be included since his usual level of intake and output were much higher than for the remaining four subjects. His patterns of excretion were exactly similar however. It will be noted that feelings of tension were associated with decreased urinary volume and urinary sodium while feelings of anxiety were associated with a diuresis of the substances, as reported by other investigators. Feelings of depression were associated with a decrease similar to that seen with tension. It is of great interest, however, that periods of active relaxation were associated with a diuresis, particularly an increase in urinary volume. Potassium excretion shows little variation with these alterations in mood, demonstrating that tubular as well as glomerular influences must have been present to produce these results.

If we re-examine the data given for excretion rates during unusual exertion or complete physical inactivity with attention to the emotional state of the individual, the marked fluctuations previously noted become understandable. Figure 2 illustrates

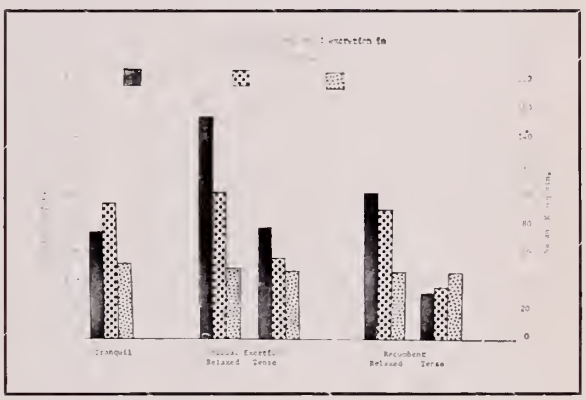


Figure 2

this well. Whether one considers the data accumulated during physical exertion or physical rest, the high excretion rates occurred when the subject felt relaxed, the low excretion rates when he felt tense.

Of equal interest is the effect of emotional states during sleep. Tension or fatigue felt at the end of the day was usually reflected in low excretion rates during the night (Figure 3). This was particularly true on those nights when the subject awakened from sleep feeling as tired and weary as he had felt the night before on going to bed. Where dreams were reported, the emotional state exemplified by the dream was often reflected in the excretion rates, suggesting that the emotion or attitude was of much longer duration than a dream is ordinarily considered to be. And where re-

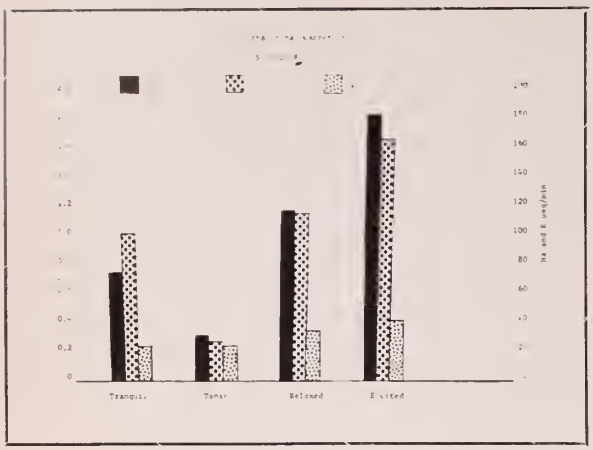


Figure 3

laxation occurred or where anxiety was present during sleep (as exemplified by dreams or by the emotional state of the subject upon retiring and awakening), excretion rates were high, exactly as had been true in the waking state.

These studies have been repeated on a metabolic ward with intake and activity carefully controlled so that the pertinent variable would be the emotional state of the individual. The frequency of stressful events and significant fluctuations in renal excretion was markedly reduced on such a ward but the associated changes in renal excretion were exactly the same (Figure 4).

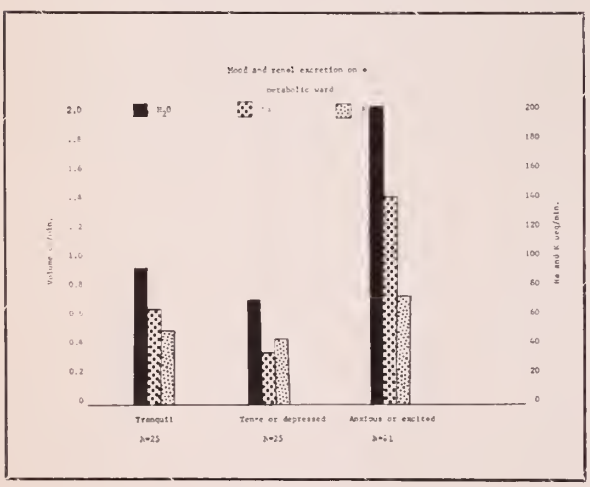


Figure 4

Again, a decrease in urinary volume and sodium excretion was observed when the subjects experienced feelings of tension or depression and a diuresis of these substances when the subject was feeling anxious, apprehensive, excited, or angry.

There are many clinical applications of these findings. The marked diuresis and loss of base which accompanies situations arousing feelings of anxiety or anger can be of major proportions. In one of our healthy subjects, an episode of intense anger was accompanied by a loss of 170 mEq. of sodium during a 5-hour period. The pertinence of this to the diabetic has been demonstrated by Hinkle, Evans, and Wolf[9] using stress interviews under controlled laboratory conditions. In one of their experiments, a diabetic subject, angered by the brusque and unsympathetic manner of her physician, excreted 1130 cc. of urine containing approximately 70 mEq. of chloride in a two hour period of observation.

There are many other clinical states in which fluid and electrolyte disturbances are important. The influence of emotional states deserves consideration in each of these. I would like to turn attention now, however, to the influence of these various emotional states on fluid and electrolyte balance in the cardiac patient who has suffered an attack of decompensation.

In 1953 Chambers and Reiser[10] presented correlations between stressful life situations and the onset of congestive failure as gained by interviewing consecutive admissions of cardiac patients. They reported two types of situations as of special importance in the precipitation of episodes of failure: 1) events which aroused feelings of rage and frustration, and 2) events which aroused feelings of rejection and loss of security. The hemodynamic changes associated with anger made the first group readily understandable to them; but they had no explanation for the second and larger group, many of whom they described as tense or depressed.

Doctor Robert Barnes has been working with us for the past year and a half to document the pertinence of emotional states and their attendant alterations in renal excretion to the status of the cardiac patient.[11] Data he has collected on one of these patients will illustrate the type of relationships which he has discovered.

This patient was a man of 43 years with hypertensive heart disease. His blood pressure as taken by his local physician remained fairly constant at a level of 210/130. His renal function, however, remained good. Intravenous pyelograms showed good renal function, his blood urea nitrogen was 18. An electrocardiogram showed no evidence of infarction or ischemia and a chest film while the patient was in failure showed a grossly enlarged heart with some enlargement of all chambers but chiefly of the left ventricle. His weight on admission to the hospital was 243 pounds, some 75 pounds above his dry weight.

During his hospital stay of 10 weeks, all 24-hour weight gains of 1½ pounds or more could be explained easily in terms of events which aroused feelings of tension or depression. On one occasion his father was hospitalized because of a stroke. He had no word about his condition for two days and gained weight during this time. On another occasion he complained that the nurses were not following their instruction properly. The resident explained that he had misunderstood and that the nurses were correct. The following day the patient apologized to each nurse who came into the room since he could not remember to whom he had made the complaint. During this 48 hour period he gained more than 4 pounds in weight. Similar events could be found accompanying each of the major periods of weight gain.

During his hospital stay he received 14 injections of mercuhydrin, 2 cc. each. Twelve of these were given during the initial 5 weeks with an accompanying weight loss of 73 pounds. His average loss in the 24 hours after each injection was 4.2 pounds but the range was from a gain of 0.8 of a pound to a loss of 7.3 pounds. All major weight losses (6 pounds or more) following mercuhydrin injections were on days where the nursing notes commented on the calmness and relaxation of the patient, not a common comment for this particular patient. Weight losses of less than one pound in the 24 hours after mercuhydrin all occurred on days when the nursing notes commented on stresses on the ward which had aroused feelings of tension or depression in the patient. The one period during which he gained 0.8 of a pound during the 24 hour period following an injection of mercuhydrin occurred on a day when another patient was moved into the room with him. This patient had been in and out of the hospital for several years, was well known and a favorite among the staff. The cardiac patient seemed to resent his presence and did everything he could to take the attention away from his new roommate. However, this occurred only a few days following the reprimand for complaining about his nursing care and during the day he told one nurse that he was afraid to complain anymore for fear of getting "kicked out."

When a stable weight seemed to have been attained, short term observations were begun. During these the patient sat quietly in his room sewing, an occupation which he enjoyed, from nine to ten in morning and from eleven to twelve. During the hour from ten to eleven a variety of observations were made. Some of these periods were spent sitting quietly in his room, some walking, others lying down, and some concentrating. One of the periods of concentration was spent sorting the cards of the Minnesota Multiphasic Personality Inventory. The others were spent on mathematics problems, since this patient had said that mathematics was particularly difficult for him.

Table 6 shows the effects on urinary excretion rates of these various activities. The patient was on a low sodium diet which accounts for the low excretion rates for this substance.

FLUID AND ELECTROLYTE EXCRETION IN A CARDIAC PATIENT

		(—Excretion—)	
		Volume	Sodium*
Conditions	N	cc/min	uEq/min
Recumbent	4	0.97	8.2
Sitting	4	0.62	2.5
Walking	4	0.40	2.4
Sitting and Concentrating	2	0.45	0.3

*Patient on a low-sodium diet

Table 6

The average excretion rates during periods of lying, sitting, and walking betwen 10:00 and 11:00 a.m. showed the decrease in urinary volume and a decrease in urinary sodium which has been reported by other observers to occur in cardiac patients when sitting or walking. It was interesting to observe one episode of sodium diuresis in this cardiac patient during an hour of walking. On this occasion he was feeling quite angry and upset about the early morning cereal which he had received. His sodium excretion rate on this occasion were more than four times the average for the other three occasion when he was walking. The most striking finding, however, was the sodium excretion observed when the patient was sitting quietly but concentrating on the Minnesota Multiphasic Personality Inventory or on mathematics. Sodium excretions at these times averaged 0.3 uEq/min., one-eighth that observed when he was sitting quietly and not concentrating.

In these experiments, the activities for the hour from ten to eleven had been determined well in advance. The patient knew each day what he would be doing during that hour. It is of considerable interest to note the variations in excretion rates between nine and ten and their correlation with what the patient was anticipating doing during the following hour. Table 7 summarizes these data. Excretion rates during the hour from nine to ten, all of which were spent sitting quietly and sewing, were quite different depending upon whether the patient was expecting to lie, sit, walk or concentrate during the subsequent hour. This difference in anticipation was the only consistent difference known to the investigators.

THE EFFECTS OF ANTICIPATION ON RENAL EXCRETION

(Patient sitting during all observations)

		(—Excretion—)	
		Volume	Sodium*
Anticipation of	N	cc/min	uEq/min
Continued sitting	5	0.53	2.1
Walking	4	0.34	1.5
Concentrating	3	0.28	1.0

*Patient on a low-sodium diet

Table 7

The mechanism by which these changes in renal excretion occur has not been adequately investigated. Table 8 shows data on endogenous creatinine excretion which indicates that glomerular influences play a role in the decreased excretion which accompanies feelings of tension and in the increased excretion which accompanies feelings anxiety or anger.[8e] It seems likely that tubular mechanisms are involved as well but these have not been investigated as yet.

CREATININE EXCRETION IN PERCENT OF MEAN OBSERVED DURING PERIODS OF TRANQUILITY

Conditions	N	Mean	S.D.
Tranquil	51	100.0	±1.9
Tense	60	84.8	±1.9
Relaxed	14	99.5	±3.6
Anxious, excited, or angry	38	110.4	±3.2
Depressed	18	90.4	±2.2

Table 8

In conclusion it should be emphasized that there is nothing mysterious or occult in the common observation that emotional reactions affect the course of a patient's illness. These responses evoke the same mechanisms which are utilized by the body to meet physical stresses. At times these mechanisms operate to maintain or to restore homeostasis, as when the diuresis of relaxation helps rid the patient with cardiac failure of excess body fluid. But these same mechanisms may, under other circumstances, lead to serious derangements of bodily economy requiring for their correction the interest and the therapeutic efforts of the physician.

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DISCUSSION

DOCTOR FREED: Doctor Barnes, a series of questions: Do you have any findings on urinary hormones in your patients? And the other question is, since many of us may not be adept in controlling the moods in our patients, is there a possibility of utilizing tranquilizing drugs to alter water and electrolyte metabolism?

DOCTOR BARNES: In answer to the first question, we did not do any of these; however, we are planning on studying aldosterone levels in reference to this problem. This may be very pertinent. We have seen in some other studies that at the time of the production of anger, a very significant rise in blood pressure of probably 30 to 40 mm. of Hg both systolic and diastolic. With this raising the blood pressure, there was an associate increase in urinary sodium. It would be very interesting to see if this is associated with alterations in aldosterone excretion.

In the American Heart Journal[1] there was an article on the diuretic effect in the tranquilizers. It would be very easy to see that the use of a tranquilizer in a patient who is very frequently discouraged and depressed could result in a change in urinary sodium, which would be beneficial. However, I don't believe that tranquilizers are necessary since a very short conversation with the patient will frequently result in these changes. One example of this was a young patient with rheumatic heart disease and an unexplained 3-day retention of sodium. The explanation of this situation came only after the patient's wife had called at the end of this 3-day period and said that "Three days ago you increased the digitalis in my husband —we both realize that his heart is very much worse now." After explaining to the patient that an increase in the dose of digitalis did not mean his heart was any worse, there was a prompt increase in the sodium in the urine.

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Role of the Connective Tissue System in Water and Electrolyte Metabolism

JOHN H. BLAND

Introduction

AN IDEA held for many years—and sometimes still held—is that connective tissue is an inert stuffing material, filling up body spaces, involved in physiology to the extent that it holds cells and organs together. Analysis of function of connective tissue is largely yet to be done.

Extravascular, extracellular space is filled with specialized cells, fibrillar elements and a complex reactive gel, whose physical and chemical properties are sensitive to its environment. Extracellular tissue space is not a void through which water and salt trickle freely. Little is known concerning transport or storage of water and dissolved solids in connective tissue. Important and provocative beginnings have appeared in the study of movement and storage of ions, water and other substances through the connective tissue barrier.[26, 27, 17, 18, 19] I'd like to review some of this data as it relates to water and electrolyte metabolism.

Role of Connective Tissue System in Water and Electrolyte Metabolism

In water and electrolyte consideration we speak as though we deal with definitive spaces—as though the body is a water bag with one or two smaller water bags inside. Are there organs or tissues as reservoirs of water and electrolyte? Does electrolyte and water accumulate in some tissues with preference to others? Most studies have dealt with muscle tissue—but muscle constitutes only 40 to 50% body weight. When measuring inulin space we measure some water space but don't know really what it is. We call it inulin space. Someone said that inulin space measures inulin space.

When normal tissue from which blood has been drained is cut no fluid escapes. The tissue looks moist. It contains more than half its weight of water. We deal with something that resembles a jelly more than a water-filled sponge.

Protoplasm has long been considered a

gel. On further consideration the extracellular supporting tissues are in the gel form.[1] It has been suggested that firmness of the gel depends mainly on magnitude of polymerization of glycoprotein. The degree of polymerization (and firmness or stiffness of the gel) varies widely from tissue to tissue—organ to organ; and at different times and different physiologic and pathologic states in the same tissue; from a tissue of the rigidity of bone to one of the softness of loose connective tissue. In rapidly growing tumors or in the symphysis pubis of pregnant guinea pigs the "ground substance" is more fluid-like, less polymerized than normal.[2] Ordinary edema, is associated with the appearance of watery fluid. Transformation from gel to sol is, however, incomplete. Clinicians record a different "feel" of edematous tissues from one case to another. Water, small molecules and ions diffuse rapidly through gels. Larger molecules such as inulin move slowly. An inulin infusion equilibrates with extracellular fluid in about five hours.[4] Hypertonic salt diffuses through extracellular space in twenty minutes.

Connective tissue is ubiquitous—present in varying forms in all tissues; a clearer picture of its capabilities, function and reactivity is emerging. While glycogen deposits accumulate in the enzymatically active interior of the living cell, most mucopolysaccharides are extracellular substances, covering cell surfaces and occurring in large amounts in intercellular spaces. Intracellular glycogen deposits change continuously during the course of many physiological processes; mucopolysaccharides are renewed at a relative slow rate. Amount and distribution of these substances remains rather constant under physiologic circumstances; their importance to the maintenance of animal life becomes apparent in disease. Research in recent years, based on chemical and histochemical methods, has shown that mucopolysaccharide production by the living cell and distribution of these substances in tissues and body fluids is altered in many states of disease and trauma.

Connective tissue fills spaces between cells and blood vessels; serves as a filter bed for nutrients, water, electrolyte and other metabolites passing to and from cells. No true interstitial "space" exists. Normally extracellular gel or ground substance fills all space between cells and groups of cells.

The Nature of Connective Tissue

Composition of connective tissue elements varies quantitatively, and in some instances qualitatively. The microscopic structure is that of cells and fibrillar components lying within a ground substance gel. Cell function depends on properties of this gel; alteration taking place within this milieu, chemical or physical, can profoundly influence nutrition and metabolism of the cell. Figure 1 lists the three basic connective tissue components.

CONNECTIVE TISSUE COMPONENTS

CELLS	EXTRACELLULAR FIBERS	MATERIAL MATRIX
1. Fibroblasts Or Fibrocytes	1. Collagen	1. Protein
2. Histiocytes	2. Reticulin	2. Polysaccharides of hyaluronic acid type and their sulfuric acid esters
3. Mast cells	3. Elastin	

Figure 1

The fundamental connective tissue system is regarded as a continuous gel matrix in which lie cells and an interlacing fabric of fibers. Parenchymal cells, bathed in matrix, may be isolated or closely set, oriented to serve a special function; renal tubules for reabsorption and secretion; skeletal muscles, nervous tissue, blood or lymph vessels with their special functions. Where connective tissue provides support only for cells, the intercellular substances consist mainly of soft matrix and reticular fibers; tendons and ligaments providing great tensile strength are composed mostly of collagenic fibers and very dense matrix-cement substance; bone and cartilage have the fibrous elements solidified by considerable amounts of cementing type (highly polymerized ?) matrix. This complex of fibers and cement substance provides a medium suitable for precipitation of calcium salts providing even more rigidity.

The nature and thickness of the connective tissue layer varies from organ to organ. In lungs and endocrine glands it is very thin; in liver and kidneys it is thicker; in heart and skeletal muscle it forms a very significant part of the organ; such is also true of intestinal tract and skin; bones, joints and tendons are formed almost entirely of connective tissue. Connective tissue is continuous throughout the body. An organ or structure is characterized by the relative amounts of the three basic connective tissue structures in its make-up, cells, fibrillar elements and ground substance.



Figure 2. Normal human connective tissue stained by silver impregnation. Gray cytoplasm and black nuclei of fibrocytes is seen. White spaces filled with fibers and ground substance.

Figure 2 illustrates normal human loose connective tissue stained by silver impregnation. The gray cellular component, fibrocytes, is seen as gray cytoplasm and black nuclei. The white spaces are filled with fibers and ground substance.

Cells: Cells of normal connective tissue are elongated fibrocytes with irregular, branching, cytoplasmic processes, building up a sponge work, the meshes of which are filled with matrix and fibers. Tissue mast cells and histiocytes may be present.

Fibrillar Elements: The three fibrillar elements are collagen, reticulin, and elastin. Collagen fibers are largest and most abundant. Collagen fibers are in bundles of parallel fibrils. Collagen is a protein. It swells more in acid solutions than any other protein. It is nearly insoluble in water but on heating is converted to a water soluble substance—gelatin.

Ground Substance: Ground substance is the extracellular, extra-fibrillar amorphous matrix of connective tissue. Certain components are probably derived from the fibroblast. These are acid mucoprotein, acid mucopolysaccharide and dispersed or soluble collagen (pro-collagen). Other components not locally derived include water, electrolytes, small organic molecules (e.g. glucose and urea) plasma proteins and cell metabolites. Ground substance is unfortunately often equated with acid mucoprotein and acid mucopolysaccharides; however on a quantitative basis and may be on a functional basis such other constituents as neutral mucoproteins, plasma proteins and soluble collagen are more important.

Many presumably distinct mucopolysaccharides of connective tissue have been identified, the principal ones are hyaluronic acid, the various chondroitin sulfuric acids and heparin. These are all polymers of high molecular weight, composed, among other moieties, of hexosamines (glucosamine for hyaluronic acid and galactosamine for chondroitin sulfate) and glucuronic acid. Figure 3 illustrates a repeating unit of chondroitin sulfate.

Metabolically mucopolysaccharides are slowly turned over. When one considers that all materials entering and leaving cells must move through the ground substance, its importance is evident. It has been sugtural elements exerting an influence on the

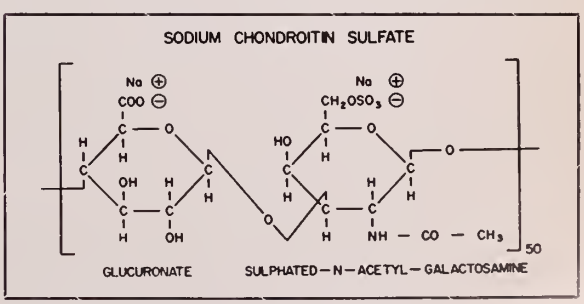


Figure 3. Note the glucuronic acid with a free anionic group capable of binding a cation to form a neutral salt. The galactosamine can likewise accept a cation. There are considered to be about fifty such repeating units in a chondroitin sulfate molecule.

gested that mucopolysaccharides function like a reactive gel, like an ion exchange resin, binding and releasing ions.[17, 18, 19, 26] Theoretically changes in concentration or state of polymerization could alter connective tissue capacity to bind inorganic ions and water. Hyaluronic acid is very hygroscopic and could exert water binding function in the tissues. Study of body fluids is incomplete without consideration of gels, that evasive and pervasive ground substance, in which water and electrolyte is in storage and transit.

It is convenient to subdivide amorphous intercellular substance into ground substance and cement substance. Ground substance is a term used for sols or soft gels and cement substance refers to more concentrated and rigid gels.

The body is commonly described as a great community of cells. It is more accurate to describe it as an edifice of intercellular substance in which the cells live as residents. Cells are jelly-like. The form of the body is due largely to its content of these stronger and non-living materials that are disposed between cells. Connective tissue not only connects the individual organism into a functional whole but as Doctor Asboe-Hansen of Copenhagen has said, "Connective tissue connects the numerous branches of medical science. Without connective tissue medicine would come to pieces, non-viable pieces, like cells of the human body would come apart without connective tissue."

Metabolic Movement Through Capillary Walls to Surrounding Connective Tissue

The capillary wall acts as a semi-permeable membrane allowing free passage of water and solutes, with a diameter below approximately 100 angstrom units. However, permeability of this barrier may increase; even cellular constitutents of blood penetrate into surrounding connective tissue.

Considerations of blood-tissue interchange place insufficient emphasis on the multiple nature of the capillary barrier. The capillary wall proper (endothelial cells and the cement substance) is only a frame work on both sides of which are superimposed structures involved in the metabolic exchange of materials across the capillary. Five distinct structural elements are encountered as water and electrolyte cross the capillary and enter surrounding connective tissue.

(A) Proteinaceous film-like component lining the capillary (? from plasma or platelets).
(B) Endothelial cells proper.
(C) Intercellular cement substance.
(D) Condensed capillary sheath.
(E) Connective tissue proper including the ground substance and its fibrillar constituents.

Movement of substances across the capillary barrier is governed by such physicochemical forces as:

(A) Concentration and hydrostatic pressures.
(B) Pore size of component structures.
(C) Thickness of each structural component.
(D) Molecular diffusion characteristics.
(E) Lipid solubility of involved materials.

The capillary surface is an extensive cellular area with less than 1% intercellular zone. Does the bulk of exchange occur across the entire capillary surface? Is it restricted to the more permeable intercellular cement?[6] Pappenheimer,[7] from studies with isotopes, suggests that small molecules, water and electrolytes diffuse in and out of the capillaries through an extremely small area of total available surface.

Inner Layer: The inner proteinaceous layer closely adherent to endothelium and cement substance, is so thin as to be usually invisible by microscopy. It seems to represent a blood constituent absorbed on to these surfaces. Its nature is not established. The evidence for its existence is indirect in nature. The concept arose from microscopic observations in mesenteric capillaries of living animals. This capillary lining is a labile entity readily altered in functional and pathological states. Zweifach[8] suggests that some edema states attributed to change in colloid osmotic pressure and protein depletion may be ascribed to alteration in the protein layer.

Experiments in perfusion of the capillary

system with colloid free mixtures have shown that the protein lining of the vessel could be "washed out"; progressive edema follows.[12] Perfusion medium manipulations (other than addition of colloid or protein) do not restore permeability. Small amounts of colloid have a restorative effect on permeability out of proportion to their osmotic pressure values. Electrolytes solutions are ineffective in restoring the protein lining.

Intercellular Cement: The basic structure of intercellular cement substance is presumed to be a dense, highly polymerized substance, probably a mucopolysaccharide protein complex. A large molecular component is superimposed on it by electrical, chemical or surface tension forces plugging most large pores, reducing overall perviousness of the structure. Pore size, number and shape of pores are thought to be determined by the physico-chemical nature of the cement and its absorbed component.

Cement substance is a labile structure with variable physico-chemical make up. It may be in a continuous state of change. The nature of the cement is affected by changes in electrolyte concentration. Calcium is essential for maintenance of normal characteristics of this material. Calcium excess seems to increase cement substance. In rat and rabbit mesentery under microscopic observation, perfusion experiments with calcium free perfusate have shown cement to go into solution and wash away.[6] Acid pH of perfusate results in dissolution and washing away of cement. Loss of cement is followed by progressive edema of surrounding tissues.[8, 23]

In tissue culture preparations endothelial growth in sheaths or tubes is dependent upon the presence of adequate amounts of calcium. If calcium content of tissue culture media is reduced the endothelial sheath begins to fall apart, cells move away from one another and round up. With re-introduction of calcium these cells again flatten out and join to form a continuous sheath or endothelial tube.[9, 10] A small amount of calcium, about 10% of normal, is enough to maintain some adhesiveness.

Absence of adhesive cement substance in calcium free media does not necessarily indicate that material is not being produced; it may be secreted in a soluble state.

It seems significant that a structural element, cement substance, constituting only about two tenths of one per cent of the entire capillary surface, represents the major factor effecting the bulk of the exchange, water and electrolytes, between blood and tissues.[7] Pappenheimer[7, 11] on the basis of hind limb perfusion studies, concluded that exchange of water and lipid insoluble materials (electrolytes) must occur across an extremely small fraction of the total capillary surface. Oxygen, carbon dioxide and lipid soluble materials probably permeate through the cell surface.

Endothelial Cells: Capillary tone or elasticity is a reflection of tone of the endothelial cell itself. This property depends considerably on the cell's water content. Injection of hypotonic solutions in tissue adjacent to capillaries causes them to swell. This may proceed to obliteration of the lumen. Micro-injection of hypertonic solutions causes endothelial cells to shrink away from one another—to the point of leakage of red cells through spaces between cells.

Zweifach[23] has by micropipette introduced silver nitrate into capillaries. This causes blackening of the intercellular cement. Excessive concentrations irreversibly damage the endothelial cell. Then no regeneration of cement material occurs. Endothelial cells may serve the important function of replacement of intercellular material; secretion is postulated.

Perivascular Sheath: The last feature of the capillary wall is the sheath of fibrils embedded in ground substance, closely applied to the outer endothelial surface. It varies in thickness and consistency in different parts of the vascular system. This condensation of connective tissue ground substance presents a barrier to water and electrolyte to and from the capillary lumen. Leukocytes and red cells under experimental conditions enter the area between endothelium and capillary sheath.

Important changes in the peri-capillary sheath occur. In scorbutic guinea pigs the sheath is readily ruptured by micromanipu-

lations with extravasation of material from blood into peri-capillary connective tissues. Two events occur in scorbutic pigs:

(1) Weakening of the perivascular sheath, probably resulting in fragility of vessels.

(2) Cement substance later becomes deficient.

Hyaluronidase introduced by a micropipette on the vessel wall results in erosion at this point and petechial hemorrhage. Hyaluronidase has no visable effect on cement substance inside the capillary. This supports the concept that cement and ground substance are two distinct chemical entities.[24] There is the impression that common forms of increased capillary fragility are due to biochemical changes in the pericapillary layer.

The importance of components of the capillary wall in water and electrolyte exchange between blood and tissues is emphasized. It has been suggested that thinness is the factor permitting the cell to act as a permeable porous barrier. This seems fallacious. There are many examples of thin layers of protoplasm maintaining a selective type of permeability. Evidence concerning endothelial cell metabolism indicates that it duplicates metabolic phenomena of other living cells. Until there is evidence to the contrary, it is reasonable to assume that endothelial cell permeability characteristics are like those of other cells. This requires explanation other than cellular permeability as the primary consideration in capillary permeation and exchange of materials from blood to tissues.

Movement of Water and Electrolyte Through Connective Tissues

Final structural component of blood-tissue exchange is connective tissue proper. Injection of diffusible dye into ground substance results in rapid, even diffusion, as when dyes are introduced into a gelatin block. Several conditions could limit free movement of water and electrolyte in this medium — or accelerate it. The gelated ground substance can be transformed toward a sol by factors to be discussed (accelerate movement water and electrolyte). Avidity for cations of both fibrous elements and ground substance may change and in turn alter avidity of these structures for water.

I'd like to raise some questions and speculate on some ideas regarding movement of water through connective tissue. Water might move between or along fibrils, in thin layers or films, so thin that water is no longer free but held to fibrils by surface forces; like a fluid film between two plates of glass; captured, unable to move freely but still a fluid, not part of the ground substance, still capable of behaving chemically as a fluid—able to transport ions and diffuse into cells. Such fluid films would be spread more rapidly along fibrils by mechanical squeezing of tissue elements as occurs with dye.

Do electrolytes move through the fluid? Or are they loosely, chemically combined, as in an exchange resin, in the ground substance? (See Figure 3.) What is the relationship of the fibrils to fluid and electrolyte transport?

Collagen can take up water and swell immensely. There are great numbers of connective tissue fibers in nearly all tissues. Millions of fibers must present an enormous surface area upon which water may exist. This could account for the fact that measured extracellular fluid volume is as large as reported, amounting to nearly 30% of all the body fluid.[13, 14]

Gaudino[15] has done some illuminating experiments relating to desoxycorticosterone and its possible influence on connective tissue and the fluid spaces. He used the techniques of inulin and heavy water (D_2O) space measurements. Figure 4 summarizes the first set of these experiments.

In dogs during DCA administration (30 mgs./day) inulin space increases progressively; D_2O space (total body water) remains approximately normal or decreases slightly; intracellular water volume decreases; interstitial space (containing the connective tissue) increases markedly; and plasma volume decreases slightly or remains unchanged. These changes tended to return to normal despite continued DCA adminis-

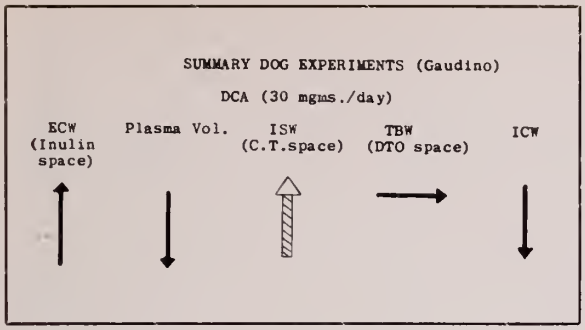


Figure 4

tration at about the 11th day. Sodium seems to move from interstitial connective tissue to an intracellular position after DCA treatment. Serum potassium fell to half the control values.

Adrenalectomized dogs were maintained on 15 mgs. of DCA per day; they showed no signs of adrenal insufficiency. Measurements of the total body water (D_2O space) and inulin space were the same as those in the normal animal receiving DCA.

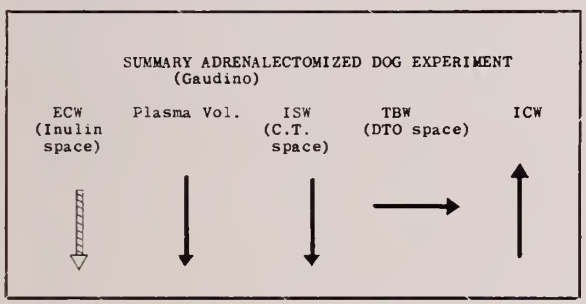


Figure 5

Figure 5 illustrates the following events: therapy was discontinued; inulin space progressively decreased but total body water did not change; inulin space declined an average of 50% from control values; total body water did not change; intracellular water increased to 30%. As inulin space declined, glomerular filtration, and PAH clearance were markedly and progressively reduced until the animals died. Blood potassium concentration increased; sodium concentration decreased; the BUN rose; specific gravity and hematocrit rose.

In these adrenalectomized animals plasma volume remained at control values during *early mild degrees of insufficiency;* changes in the other compartments were common. It appeared as if in early adrenal insufficiency plasma volume and intracellular volume were maintained at the expense of interstitial space.

The experiments suggest that DCA has an influence on maintaining and expanding interstitial space (containing connective tissue) possibly by some as yet unidentified influence on connective tissue fibrils or matrix. DCA stimulates collagen formation which may be a factor.

The question of capability of the ground substance or collagen to bind water remains unanswered. There surely are degrees of water binding in interstitial space. Bound water, has been defined as water which though fixed in place, can be molecularily exchanged. The term "bound water" should be reserved for water unavailable as a solvent and not considered in measuring activity or osmotic concentration. Forces binding water are unknown.

Doctor Karl Meyer postulates that fibrous structures are hydrated; that the structures are colloids and that water phase will bind certain ions without serving as water of solution; that it will bind sodium, chloride and potassium in readily exchangeable form.

From these considerations, it appears that an important and little known function of connective tissue is that of being a buffer compartment, a depot; an avenue of exchange for water, electrolytes and other substances necessary for cell metabolism or resulting from it. If interstitial space is composed mainly of connective tissue this would clearly appear to be one of the main functions of connective tissue.

Ground Substance as Polyelectrolyte*

Ground substance of connective tissue contains three components:

(1) Colloids

(2) Diffusible electrolytes

(3) Water

The polyelectrolyte nature of the anionic polysaccharides of connective tissue sug-

*A highly aggregated, high molecular weight molecule with varying anionic or cationic charge density, capable of binding cation or anion depending on its charge, e.g. Chondroitin sulfate, a salt of molecular weight 20,000 to 30,000, often isolated with sodium or potassium as its cation: polymer consists of about 50 repeating units, which, if completely ionized could carry a total negative charge of 100 units per molecule. As dry neutral salt, this charge is balanced by 100 equivalents of cation, sodium or potassium.

gests that they may bind cations. Since hyaluronic acid and the chondroitin sulfates constitute such important components of connective tissue, the binding properties of these polyelectrolytes may influence distribution and storage of extracellular cations. Furthermore changes in extracellular polysaccharide (e.g. depolymerization) metabolism might influence the course of diseases in which storage and distribution of sodium play a major role.

The amounts of water and electrolyte in the ground substance depends on its electrochemical properties. One measurable property is the charge density of cation or anion binding groups of immobile colloids. This quantity may be estimated as equivalents per kilogram of tissue water. Catchpole, Engel and Joseph[17,18,19] measured electrical charge density using the liquid junction potential method. They further measured the effect of certain hormones on interaction of sodium, potassium, calcium and magnesium in living connective tissues.

In normal pubic symphysis of guinea pig, the charge density is high and water content low. In the relaxed (?depolymerized) symphysis pubis of the pregnant guinea pig, the charge density is low and the water content very high. Mobility of potassium is very high in relaxed state. Interaction of the negatively charged ground substance (chondroitin sulfuric acid) with sodium and potassium favors selective absorption of potassium in preference to sodium.

Calcium content of tissues other than bone is related to the density of the negatively charged ground substance of the tissue. Highest values for ionic and bound calcium are found in dense connective tissue. Hormones act on connective tissue to vary the state of ground substance aggregation; it is expected that this will affect distribution of calcium, which it seems to do.[17] There is a high affinity between calcium and chondroitin sulfuric acid. Dense tissues, e.g. cartilage, contain ionic calcium in concentrations three times that of blood while bound calcium concentration exceeds blood values by 10 times.[17]

The total potassium content of connective tissues, like the total calcium, depends on the state of aggregation of the matrix, colloids. Loose connective tissue with high water content, containing small amounts of insoluble highly aggregated colloid have potassium and calcium concentration approaching that of blood; but in cartilage, potassium concentration is more than ten times that of blood and total calcium is concentrated almost to the same extent.[17,18] Small increases in blood potassium seemed to result in increases in ground substance potassium, possible displacing sodium and calcium. At any rate inorganic composition of ground substance was responsive with varying sensitivity to changes in blood electrolytes—those that can enter the matrix and react with colloid.

Current theory suggests that glycoprotein disaggregation and formation of water soluble fractions are features of hormone action on connective tissues. Potassium is immobilized by dense connective tissues which thus act as ion exchange resin for potassium. In loose connective tissue, or in those loosened by hormonal action, colloidal charge density is decreased. Consequently relative mobility of potassium as compared with sodium increases from its low value in dense tissues to much greater mobility in loose tissues, the latter approaching its mobility in water. Thermodynamic properties of ground substance show a strong tendency for potassium to displace sodium from the colloidal phase.

It is apparent that connective tissue components, ground substance and fibers are involved in tissue permeation and in water-electrolyte, acid-base and osmotic equilibria. Interestingly, Schade[20] in 1923 published a paper proposing a theory similar to Catchpole's modern one. He formulated a regulatory role for collagen fibers and ground substance in water and electrolyte metabolism. He showed specific ions to have some influence on the colloidal state of connective tissue. Increase in sodium concentration was accomplished by increased colloid dispersion and water retention; accumulation of potassium or calcium was followed by shrinkage and loss of fluid.

Hormone Action on Connective Tissue

Organ and tissue responses to hormone action shows evidence of connective tissue participation.

The following hormonal effects on connective tissue have been observed: The hormones cortisone, hydrocortisone and corticosterone act on all components of connective tissues, cells, fibrils and ground substance.[28] Fibroblasts are reduced in number.[28, 29] Collagen production is reduced.[28] Permeability of ground substance is decreased.[30]

Growth hormone increases fibroblast proliferation and formation of collagen fibrils.[31] Granulation tissue becomes abundant. Collagen formation in skin and muscle increases.[32]

Thyrotropin has a direct effect on connective tissue.[33] Injection of thyrotropic hormone causes accumulation of water binding hyaluronic acid in retrobulbar tissue and consequent exophthalmos.[33, 34] Injection of thyrotrophic hormone in the guinea pig is rapidly followed by a transport of fat from normal depots to muscles, liver and kidney.[33] Mucopolysaccharide accumulation has been experimentally demonstrated in peripheral muscles, in the peri-renal and axillary regions.[33] These changes occur in intact as well as thyroidectomized animals. Thyroxin inhibits thyroid stimulating and exophthalmic effect as well as the fat mobilizing effect of thyrotrophin. The effect of thyrotrophin on connective tissue seems opposite to that of cortisone. Cortisone exerts a distinctly inhibitory effect on connective tissue elements, cells as well as ground substance; thyrotrophin has a stimulating effect with increase in hyaluronic acid.

Thyroxin has no known effect on connective tissue.

Estrogens influence the connective tissue with most emphasis on the ground substance, inhibiting permeation of electrolyte, water and dyes. Intercellular fluid is increased, presumably due to increased formation of hyaluronic acid though this remains unproved.[35] Women often become slightly edematous immediately before a menstrual period. At this juncture estrogen excretion in the urine increases. Weight is gained and the daily output of urine is decreased for a few days, consistent at least with water retention.

Testosterone renders connective tissue looser and less compact. Collagen bundles become separated by increased amounts of matrix. This material is hyaluronic acid (experiments on cock's comb).[36]

Many endocrine diseases involve change in mesenchymal tissues directly, e.g. myxedema, thyrotoxicosis, malignant exophthalmos, adrenal insufficiency, acromegaly, Simmond's disease and Cushing's disease. Connective tissues have been clinically considered important in endocrine diseases as those noted and the obvious rheumatic diseases. The fundamental changes here described suggest that connective tissue is a reactive, actively metabolizing tissue, playing an important part in water and electrolyte regulation. The binding of cations by the anionically charged connective tissue polysaccharides probably has physiologic importance. There is good reason to implicate connective tissue constitutents as possible areas of storage of osmotically inactive sodium in certain disease states in man.

It is the ambition of this presentation to suggest an attitude toward and interpretation of the little available information on the metabolic relationship between connective tissue, water and electrolyte. The stuffing is alive.

In studying and contemplating such delightfully slimy substances as hyaluronate and chondroitin sulfate I am reminded of a wonderful poem by Rupert Brooke. It is called Heaven. Essentially it is a fish's prayer. As one reads the poem he visualizes a fish, replete with flies, lolling away his noon hour on the bottom of his brook. He is glorying in the marvelous mud, water and slime all about him. He contemplates his long range future—is there a great by-and-by for fish? Surely there is an even better world Beyond. Water and slime are such wonderful things for fish and men. I'd like to quote a few lines.

> One may not doubt that, somehow, Good
> Shall come of water and of mud;
> And, sure, the reverent eye must see
> A purpose in Liquidity.
> But somewhere, beyond Space and Time,
> Is wetter water, slimier slime!

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The Metabolic Response To Injury

CURTIS P. ARTZ

INJURY, irrespective of type, initiates a series of physiologic events, effecting every organ and probably every cell in the body. In general, the magnitude of the metabolic derangements is in direct proportion to the extent of the stimulating injury. The response of the organ systems of the body is a continuing one and may cover a period of days or weeks in accordance with the type and magnitude of the trauma and the state of the individual at the time of injury.

Injury, whether it may be a surgical operation, mechanical trauma, or a burn, stimulates the central nervous system and the adrenal medulla (Figure 1). The adrenal

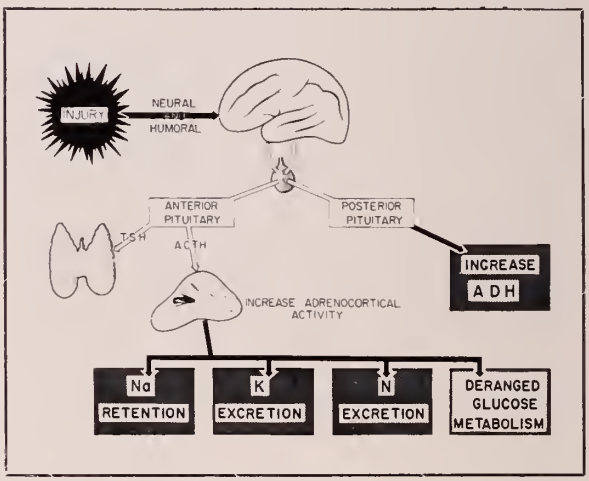


Figure 1. Schematic outline of neuro-endocrine changes and the resultant derangements occuring after injury.

medulla secretes increased amounts of epinephrine and norepinephrine. Stimuli from the central nervous system and the adrenal medulla effect the hypothalamus, which in turn stimulates both the anterior and the posterior pituitary. The anterior pituitary releases two important hormones, adrenocorticotrophic hormone (ACTH) and thyroid-stimulating hormone (TSH). The posterior pituitary secretes an increased amount of antidiuretic hormone (ADH).

Although several mechanisms may be involved in the stimulation of the adrenal cortex, the degree of the response is largely dependent upon the rate of release of

ACTH by the anterior pituitary. With this release of increased ACTH soon after injury, there is a consequent increase in adrenocortical hormone excretion. There is a prompt rise of 17-hydroxycorticosteroids in the plasma. The 17-ketosteroid excretion in the urine rises but soon falls. The 11-oxycorticoids are increased in the urine, and this increase may persist for several days. Typical responses after various types of injury are shown in Figure 2. After gas-

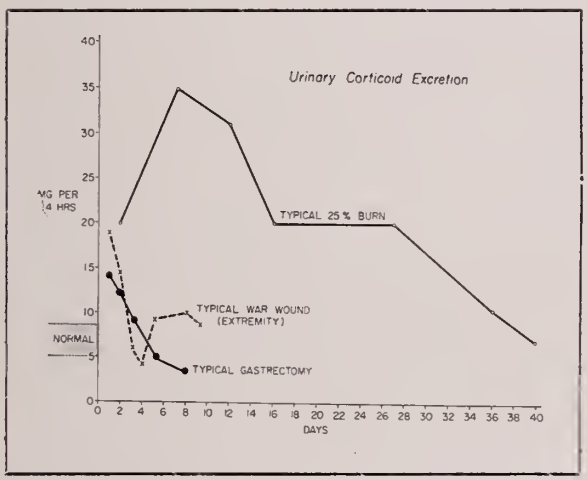


Figure 2. Urinary corticoid excretion in typical cases of various types of injury. The curves of the 25% burn and the gastrectomy were plotted from data of References 2 and 3. The curve of the typical war wound was plotted from data in Reference 1.

trectomy there is an immediate rise in the urinary corticord excretion, which usually falls to normal in six or seven days. In more severely injured patients such as those with large war wounds of the extremities, there is a more pronounced rise immediately after injury, which falls to normal after eight or ten days, unless there are secondary operative procedures or retained dead tissue. The rise in 11-oxycorticoid excretion in burns is more marked. There may be considerable increase in excretion in the first eight or ten days, which then diminishes for a period, but does not reach the normal range until well along in the convalescent period after the 35th or 40th day. When infection supervenes, urinary corticord excretion is increased and may show an increase for prolonged periods of time.[1,2,3,4]

The release of increased amounts of adrenocortical hormones and antidiuretic hormone have a pronounced effect on the sodium, potassium and water balance, as well as protein metabolism. The various adrenocortical hormones promote sodium retention, increased potassium excretion, increased nitrogen excretion and deranged carbohydrate metabolism. After injury there is a prompt antidiuretic effect mediated through the increased secretion of ADH by the posterior pituitary. There is an increased secretion of thyroxin by the thyroid gland, presumably due to increased pituitary output of thyrotropic hormone. The oxygen consumption and metabolic rate of an individual after trauma are elevated. This elevation may continue until wound healing is almost complete. One of the clinical corollaries of this increased metabolic activity occurs in burns. The pulse rate of burned patients remains above 100 until the wound is almost completely grafted. This persistence in increased pulse rate is a very consistant finding and probably represents a manifestation of increased metabolic activity until the wound is closed.

Electrolyte and Water Metabolism

Abrupt disturbances in electrolyte and water metabolism occur immediately after injury. Changes in the distribution of water occur by the early migration of fluid into and around the injured area. There is an increase in the interstitial space. Serum sodium and chloride concentrations drop immediately after injury, as there is a shift of sodium into the injured cells with an outward shift of potassium from the cells. With the expansion of the interstitial space, there is dilution that also causes a fall in serum sodium. It is well recognized that a mild hyponatremia is associated with a favorable response in severely injured patients. Sodium excretion is low initially resulting in an early positive balance, but later during the period of redistribution of fluid and resorption of wound edema, the balance becomes negative. Potassium balance is usually negative in the few days following injury, but positive balance is gradually obtained as potassium intake is increased. Generally the periods of sodium and potassium changes are considerably shorter than changes in protein metabolism. The volume

of sweat secreted is often reduced during the early postoperative period, and the electrolyte concentrations in the sweat vary in the same direction as do those in the urine. Sodium and chloride are diminished, and potassium increased. These alterations also occur in saliva.

The cumulative sodium balances in two types of injured patients are shown in Figure 3. There was a marked accumulation of sodium in the 45% burn during the early days after injury. This positive accumulation of sodium continued for eight or nine days, then as redistribution of fluid and an outpouring of water and sodium occurred and the cumulative sodium balance became negative. In the patient with the bilateral below-knee amputation there was a slow accumulation of sodium which continued for ten days, at which time a secondary operation undoubtedly was the stimulus for continued sodium accumulation.[5,6]

The cumulative potassium balances of the two patients shown in Figure 3 are plotted in Figure 4. In both the 45% burn and the bilateral below-knee amputation there was marked negativity of potassium balance during the first 12 days after injury. As more potassium was given, the cumulative balance approached zero.[5,6]

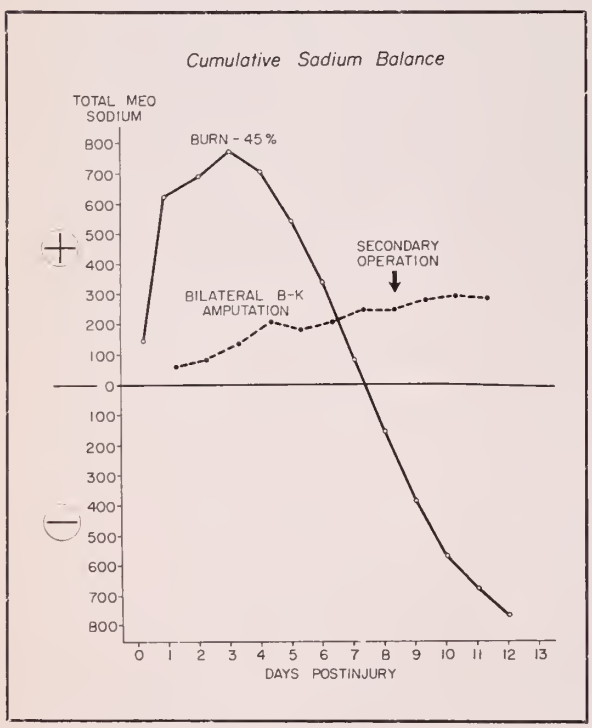


Figure 3. Cumulative sodium balance of a 45% burn and a bilateral B K amputation. There was a positive balance in the burn soon after injury which became negative after several days. The positive balance seen in the amputation probably would have fallen to zero had it not been for the additional stimulation of the second operation.

During the early postinjury phase, the outpouring of potassium may be correlated well with nitrogen losses but in some patients the early urinary potassium losses may far exceed the amount expected from determination of nitrogen losses. At the end of the first week potassium retention usually follows potassium intake. At this time potassium and nitrogen balances are completely disassociated. The daily potassium balance becomes positive, and the nitrogen balance is almost always negative. After the first two weeks the level of potassium intake has little influence on the potassium balance. Potassium balance usually becomes negative and remains slightly negative until the nitrogen balance becomes positive.[7]

The average fluid intake and urine output in 24 severely burned patients are plotted in Figure 5. The fluid intake is usually quite high immediately after injury and falls after the second day. The urinary output is quite low at first and then increases until the excess amount of water that has accumulated has been excreted. If it is assumed that the daily insensible water loss is approximately 2000 cc., and this is added to the urinary losses, the output of water will equal the intake about the third or fourth day, and eventually will exceed the intake of water on the tenth or eleventh day. In less severe burns diuresis occurs much earlier.

The daily water balance (non-cumulative without correction for insensible loss) in a severely injured patient is shown in Figure 6.[1] A greater intake than output is seen in the first few days after injury. Later a diuresis occurs with output being considerably greater than intake. The diuresis may start on the third or fourth postinjury day in moderate injuries, or take place slowly from the eighth to twelfth day in more severe forms of trauma, particularly burns.

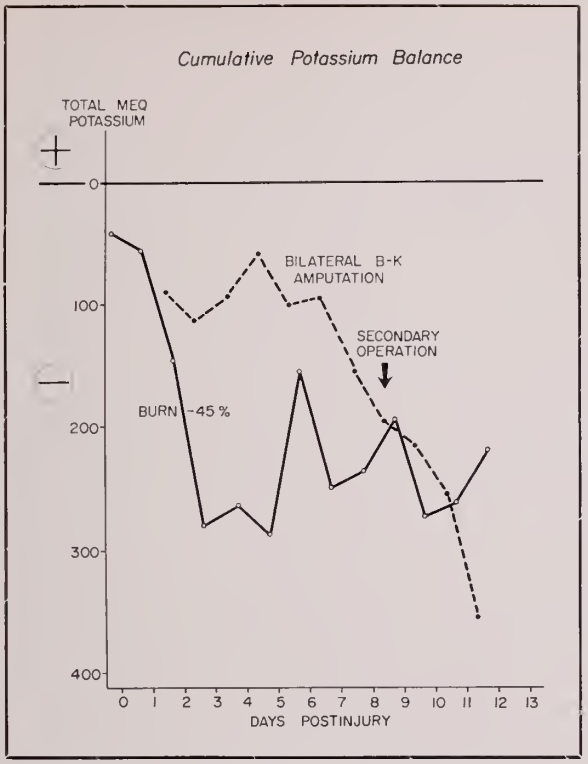


Figure 4. Cumulative potassium balance of the same two patients in Figure 3. The patient with the 45% burn, of which about one-half was third degree, lost considerable quantities of potassium. He had a marked negative cumulative balance for several days. The patient with the amputation had a less severe deficit of potassium which increased after the second operation. These accumulated potassium deficits occurred in spite of potassium therapy. There was no fall in the serum potassium level.

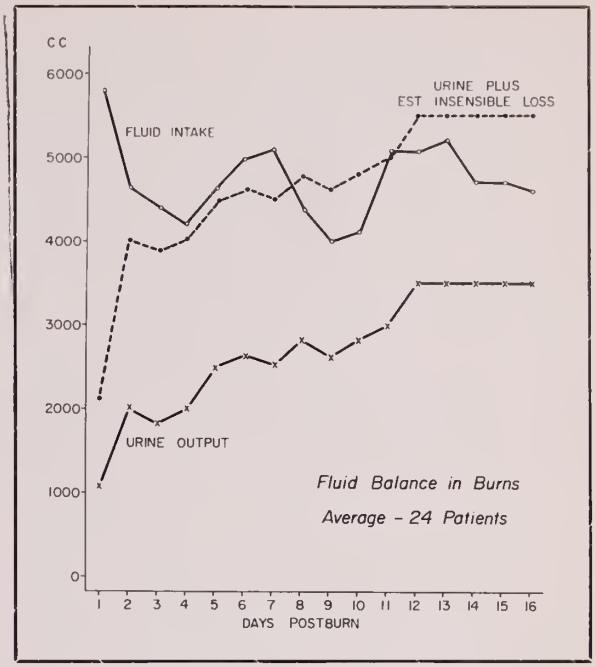


Figure 5. Chart of fluid intake and urinary output in twenty-four burned patients. The average intake and urinary output was computed on twenty-four patients with moderately severe burns. The urinary output rose very slowly over the first twelve-day period. There was no abrupt and sudden diuresis. The dotted line shows the total losses of water when insensible water loss was estimated at two liters a day. The output from urine and insensible loss was about the same as intake by the fourth day. Up until that time there was a considerable positive water balance. After the eleventh day the estimated output exceeded the intake and the expanded interstitial space diminished.

Clinical Implications. Increased amounts of fluid are required soon after trauma for supportive therapy. The plasma volume must be maintained and during this period fluid shifts into the interstitial space. This is more marked in patients with abdominal injuries and burns, than in patients with extremity injuries. Fluid does accumulate around the injured soft tissue wound, as is evidenced by edema. During this initial phase the patient will gain weight. A typical weight curve in a burned patient is shown in Figure 7.[8] After the first week the accumulated water in the interstitial space was excreted, and the patient's weight returned to normal, then due to loss of lean body mass continued to fall. The rapid early weight gain is a reflection in water gain and obscures the fact that the decreases in fat and muscle mass occur simultaneously. Early after injury fluid must be given, and usually the amount is regulated by urinary output and extra-renal fluid losses. After the first few days a diuresis is expected, and the amount of fluid intake should be determined primarily by extra-renal losses, particularly gastrointestinal losses and insensible water loss. Patients with fevers have an increased insensible water loss and require more water.

Because of the expanding interstitial space sodium must be given to burns and patients with severe injuries. There is a marked increase in the accumulation of sodium in and around the injured area. Little sodium is excreted early, and therefore, the amount of sodium given should be determined by the losses surrounding the injury. Losses from the gastrointestinal tract and into a burn site may be appreciable; where-

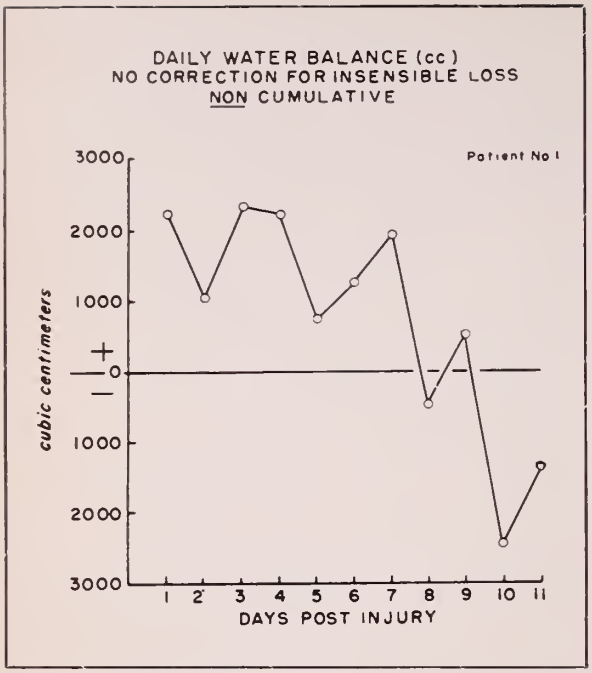


Figure 6. Water balance on a soldier who suffered a traumatic amputation of both legs after a land mine explosion. The intake of water exceeded the urinary output during the first seven days. However, no correction was made for insensible loss. After eight days the urinary output exceeded the intake. This was apparently the period of diuresis. Reprinted with permission of the author[1] and the Annals of Surgery.

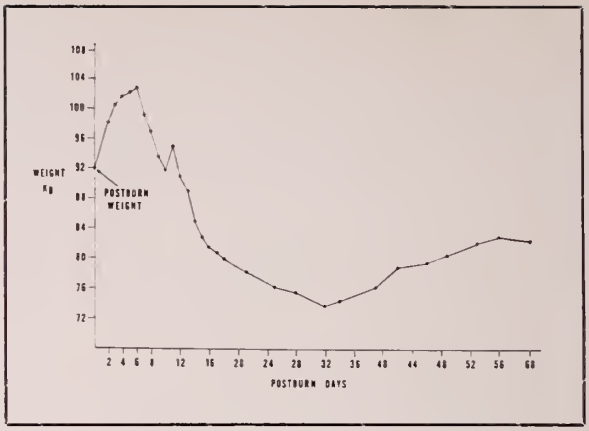


Figure 7. Characteristic weight curve of a severely burned patient. The rapid early weight gain is the reflection of water gain and obscures the fact that decreases in fat and muscle mass occur. Immediately following the maximum weight the early descending limb of the curve usually has a more gentle slope than the ascending limb preceding the maximum. This is due to delay in the excretion of edema fluid. There was a continued loss in weight with loss of nitrogen and fat until the thirty-second day. Thereafter the patient began to store nitrogen and his weight gain increased. However, the fat gain phase did not begin until after the sixtieth day.

as, early losses after uncomplicated surgical procedures are minimal and sodium is contraindicated. Burned patients require little or no sodium after the first 48 hours. In gastrointestinal injuries with gastric suction sodium may be required for several days. The serum sodium changes occurring in 25 patients with severe war wounds are shown in Table 1. In spite of the infusion of one liter of physiologic saline solution daily during the first week the serum sodium remained at low levels. A high percentage of the patients had a serum sodium of less than 130 mEq. per liter.[5] Likewise, in burns the serum sodium will fall to 130 or 135 mEq. per liter in spite of a very positive sodium balance.[6] Unless the concentration of sodium in the serum is abnormally low early after injury, it need not be corrected because a diminished serum sodium is almost invariably associated with a good response. As fluid redistribution occurs after injury, the serum sodium concentration will rise.

Hematocrit determinations after injury in battle casualties are shown in Table 2. Trauma to the extremities is usually followed by a falling hematocrit because of the losses of red cells into the injured soft tis-

HEMATOCRIT DETERMINATIONS IN WAR INJURED.[9]

Average—20 Casualties

TYPE OF INJURY	IMMEDIATELY AFTER INITIAL OPERATION	DAYS POSTOP 1	2	3	4	5	6
ABDOMINAL INJURY	50	52	47.5	48	45	43	44
EXTREMITY INJURY	40.5	38.5	36	32	33	—	—

TABLE 1.

SERUM SODIUM DETERMINATIONS AFTER WAR INJURIES.[5]

Average—25 Battle Casualties

Time	Average Serum Na	Patients With Serum Na Below 130 mEq/L
Before Operation	141	0%
0-12 Hours Post-Op	141	11%
12-24 hours	133	45%
2nd day	129	45%
3rd day	129	44%
4th day	125	46%
5th day	131	25%
6th day	131	46%
7th day	133	20%

TABLE 2.

sues. The losses of cells exceed the losses of plasma. Patients with gastrointestinal injuries usually have a rising hematocrit because the loss of plasma into the intestinal wall and peritoneal cavity is appreciable.[9] The rising hematocrit is associated with the loss of sodium and water into the peritoneal cavity. This phenomenon is somewhat comparable to that seen in burns, namely a rising hematocrit occurs because of a loss of plasma into and around the injured site.

Because of the retention of water and the release of large quantities of potassium into injured cells, there is little need for potassium immediately after injury. As the urinary output increases and renal function is assured, the increased excretion of potassium will lead to a severe negative potassium balance, unless potassium is given. Most injured patients should be given potassium either orally or parenterally beginning 48 hours after injury unless there is interference with renal function.

Nitrogen Metabolism

One gram of nitrogen represents 6.25 grams of protein. An individual lying quietly in bed requires daily about one gram of protein and approximately 30 calories per kilogram of body weight to stay in positive balance. The young healthy adult male in nitrogen equilibrium consumes approximately 60 to 100 grams of protein per day (10 to 15 grams nitrogen) and 2500 to 3000 calories. He excretes about 9 to 12 grams of nitrogen in the urine and 1 to 2 grams of nitrogen in the feces. Following trauma the fecal nitrogen is unchanged, but the urinary nitrogen increases considerably. After severe injuries, particularly in extensive burns, 30 to 40 grams of nitrogen may be excreted a day through the urine.

The chief nitrogen component in the urine is urea; proteinuria is rarely seen in the absence of renal disease. Increased urinary nitrogen excretion begins soon after injury and may continue for very prolonged periods in severe injuries and particularly in burns. During this time the injured patient is in negative nitrogen balance, and there is a gradual depletion of his body protein. Thereafter, as the patient recovers, the urinary nitrogen excretion falls, the patient's intake increases, and nitrogen balance becomes positive. This continues until the body tissues are restored.

The magnitude of urinary nitrogen excretion varies greatly. It cannot be predicted in an individual patient, but a few generalizations seem warranted:

a) There is a rough correlation between the severity of injury and the amount of nitrogen excreted; the more extensive the injury, the larger the nitrogen excretion.

b) Men generally excrete more than women.

c) Usually the urinary nitrogen excretion is less in patients who were malnourished before injury.

In addition to the losses through the urine considerable nitrogen loss may occur by way of the wound exudate. This is particularly common in burns. The amount of nitrogen lost by this route is highly variable but may account for a large fraction of the total nitrogen losses. In a second degree burn, for instance, the nitrogen exudate tends to clines rapidly as the wound heals. In third be high in the early postburn period but dedegree burns with extensive granulating wounds exudate nitrogen is well correlated with the amount of suppuration. The loss of 5 to 7 grams of nitrogen in the exudate

wherever there is inflammation, swelling, pain

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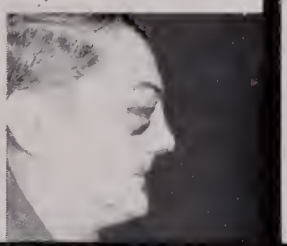
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FORCE INJURY

severe bruises . . . swelling . . . cleared by fifth day[2]





VARICOSE ULCER

15 years duration . . . resolved with VARIDASE[1]





INFLAMMATORY DERMATOSIS

rapidly spreading rhus dermatitis healed within a week[1]





INFECTED LACERATION

marked reversal in 3 days . . . returned to school . . . closure advanced[1]





THROMBOPHLEBITIS

back on his feet in a week after recurrent episode[1]





REFRACTORY CELLULITIS

normal routine resumed after 4 days of VARIDASE[1]



per day is not uncommon from infected granulating surfaces.

The direct duration and magnitude of negative nitrogen balance are influenced by the nutritional regimen used as well as by the severity of the injury. An energetic nutritional regimen, instituted 7 to 10 days after injury, may reduce substantially the duration of negative nitrogen balance. Unless specific efforts are made to achieve ingestion of a high protein, high caloric diet, negative balance may last for a prolonged period.

Since burns are the most severe form of injury, the magnitude of changes in nitrogen balance are more marked and for longer periods of time than in most other injured patients. Nitrogen balance has been more intensively studied in burns than in other injuries. The cumulative nitrogen balance of a 70% second degree burned patient is shown in Figure 8. This healthy 98 kilogram young male was in negative nitrogen balance in spite of an appreciable intake for 28 days after the burn when cumulative loss of nitrogen was 382 grams and his weight 74 kilograms.[5] As most of the second degree burn healed and the wound closed, he was able to achieve positive nitrogen balance, and therefore many of the cumulative losses were replaced. His cumulative nitrogen balance did not return to zero until the 56th day after injury. At that time he began storing additional nitrogen. His weight had increased but not returned to normal because the fat losses had not been replaced. The last phase in the metabolic response to injury is the so-called "fat-gain phase."[10]

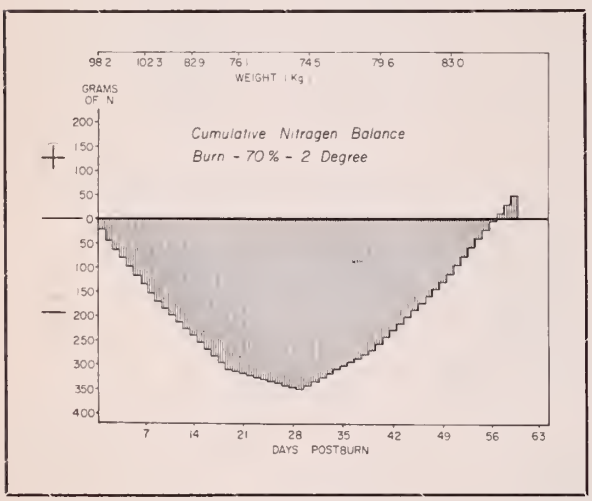


Figure 8. Cumulative nitrogen balance of a patient with a 2nd degree burn of 70% of the body surface. This is the same patient whose weight curve is shown in Figure 7. There was a continuing loss of nitrogen over and above intake for 28 days. During that time the patient lost from 92 to 74 kilograms. Thereafter the balance became positive and the cumulative losses of nitrogen decreased. At the fifty-sixth day the deficit had been replaced and part of the weight loss had been recovered.

The changes in serum proteins following injury are essentially the same as those changes following elective operative procedures, except quantitatively they are more pronounced. Likewise, the changes are even more pronounced in the very severe burn. Following abdominal injury there is a rapid decrease in the albumin-globulin ratio, followed by continued progressive decrease for the next few days. Patients with extremity wounds show a delayed but progressive decrease in albumin-globulin ratio. Electrophoretic studies reveal the changes to involve primarily a decrease in the relative per cent albumin and an increase in the relative per cent of alpha[1] and alpha[2] globulin fractions.[11] Burns of 10 to 15 per cent of the body surface, unless they are very deep, show no marked changed in the level of serum protein, albumin or globulin. In more extensive and deeper burns, however, there are marked changes. Immediately after burning there is a sharp reduction in total serum protein level. This may reflect the expanded interstitial space, which has occurred during fluid and electrolyte administration immediately after the burn injury. From the second to the fifth day the decreased level of albumin is almost wholely responsible for the low total protein. This low level of albumin usually remains for two months or more following the burn. There is an increase in the alpha[2] globulin after burning, but no radical change in beta globulin.[12] The serum gamma globulin during the first six days following the burn suggests only a tendency toward abnormally low levels and not an unequivocal depression. This may be a reflection of increased fluid and electrolyte therapy and expansion of the interstitial space. The initial reduction of gamma globulin is followed by an

increase above the normal range at about the second week following the burn.

The exact role of adrenocortical hormones in nitrogen metabolism after injury is not known. The effect of hydrocortisone-like steroids on nitrogen metabolism is very complex. In general the administration of ACTH, cortisone, and hydrocortisone in large doses induces increased nitrogen excretion. The exact correlation between nitrogen balance and steroid excretion is difficult to interpret. Certain conclusions seem justifiable: (a) severe burns are characterized metabolically by profound alterations in nitrogen balance and steroid metabolism, and (b) since steroids are known to influence protein metabolism, it is quite possible that alterations in nitrogen balance and steroid excretion are related casually.

It appears that the increases in urinary nitrogen excretion soon after injury are directly related to the increase in the secretion of adrenocortical hormones. The marked negative nitrogen balance is also enhanced by the absence of nitrogen intake after injury. Later nitrogen excretion diminishes. This decrease in nitrogen excretion probably reflects a decline in catabolic intracellular protein destruction and indicates that the patient is metabolically prepared to begin anabolic processes and load nitrogen. The storage of nitrogen will not be affected, however, unless adequate amounts of nitrogen and calories are supplied. This can be accomplished by way of diet, or in patients who are unable to take food orally, by parenteral solutions. The storage of nitrogen increases very rapidly after the wound is healed. As nitrogen losses are replaced and the lean body mass increases, then the "fat-gain phase" begins. Body weight increases progressively in the weeks and months that follow if adequate caloric intake is provided.[13] Burned patients will frequently gain from one-half to one pound a day during the first few weeks after discharge from the hospital.

Therapeutic Implications. Immediately after severe injury it is impossible to administer adequate proteins and calories to maintain positive balance. It is not definitely known whether an adequate intake of proteins and calories is absolutely essential. Negative nitrogen balance soon after injury is an acceptable sequel of injury and if it is not prolonged, seems to have little detrimental effect. Prolonged inability to receive proteins and calories, however, will lead to unusual nitrogen losses and severe protein depletion with the attendant predisposition to complications.

Proteins, calories, and vitamins should be given as soon after injury as is practical. In most patients a diet can be started on the second to the fourth day. If this is impossible, the infusion of a protein hydrolysate solution and glucose solutions, along with potassium, should be started. Nitrogen storage will not occur unless adequate potassium is available. Injured patients will usually require 1½ times the normal requirements of proteins and calories. In more severe injuries, particularly burns, larger quantities may be necessary. In children, for instance, about 1½ to 2 times the normal protein and caloric requirements are needed. Male patients show greater metabolic disturbances than females and require 3 to 4 times the normal proteins and twice the normal calories. Female patients can probably be kept in balance with lesser amounts. A good rule of theumb for extensive burns is 2.5 grams of protein and 60 calories per kilogram of body weight per day.[7]

The best type of feeding is a high protein, high vitamin, high caloric diet given three times a day with supplemental protein between-meal feedings. In some instances it is necessary to give supplemental protein by the use of intravenous protein hydrolysate solutions. Protein hydrolysate combined with glucose and alcohol offers 37.5 grams of protein and 800 calories per liter. This solution is a good supplemental protein product, and in patients who are unable to take food by mouth, two liters a day may be very helpful in decreasing the negativity of nitrogen balance. Sometimes when the gastrointestinal tract can tolerate food, but the patient will not eat, tube feedings are beneficial.

There is evidence that testosterone and testosterone-like hormones increase the stor-

age of nitrogen but quantitatively this is not appreciable. Increasing the intake is a better and less expensive method of enhancing nitrogen balance. Androgenic hormones have not been widely accepted for prolonged use to increase the storage of nitrogen.

It is important that an adequate red cell mass be maintained throughout recovery from injury. The hematocrit should be determined periodically and adequate blood given to maintain a hematocrit of 40 to 45.

Carbohydrate Metabolism

There appears to be a decrease in glucose tolerance and some resistance to insulin after injury. These changes reflect an increase in gluconeogenesis and in peripheral carbohydrate breakdown. The increased activity of the adrenal cortex effects carbohydrate metabolism, but this influence is not as clear as the effect of adrenocortical hormones on protein metabolism. After injury the glucose tolerance curve becomes diabetic in type, and the insulin tolerance curve demonstrates a flattening in its pattern. Both the decreased glucose tolerance and the resistance to insulin appear proportional to the degree of injury (Figure 9).[14] These abnormalities diminish as convalescence progresses.

A moderate transient hyperglycemia is a common sequel of burning. This phenomenon is of little clinical importance and usually disappears in several days. A syndrome of so-called "stress diabetes" has been observed a few times in burns.[15] Such patients appear very ill with deficits of salt and water. The phenomenon is usually associated with forced feeding. It would appear that excess carbohydrate intake in patients whose carbohydrate tolerance has been impaired by increased adrenocortical activity gives rise to diabetes. It may be that such patients are borderline diabetics before injury, and that increased adrenocortical stimulation activates a latent diabetic condition.

Fat Metabolism

There is an increased turnover of body fat after injury. A rapid loss of body fat has been demonstrated, presumably due to increased oxidation during the early post-injury period. Extraordinarily large fat losses may occur, particularly after burns. The magnitude of the estimated losses depends upon the severity of the injury. In a severely burned patient the early fat loss may average as much as 600 grams per day. Minimal fat losses have been seen in smaller burns and less severe injuries. The amount of body fat metabolized depends on the nutritional regimen used. An energetic feeding regimen tends to diminish losses. It is doubtful, however, whether any reasonable feeding program can completely prevent them. Fat losses do appear to diminish or almost cease when nitrogen balance becomes positive. After the patient is well into convalescence, he starts into a so-called "fat-gain phase." This phase usually follows repletion of protein deficit, healing of the wounds and ambulation. With a high caloric intake it is sometimes possible to see the deposition of fat, particularly in the face and around the waistline. With additional muscular activity and exercise there occurs a general redistribution of this fat later in convalescence.

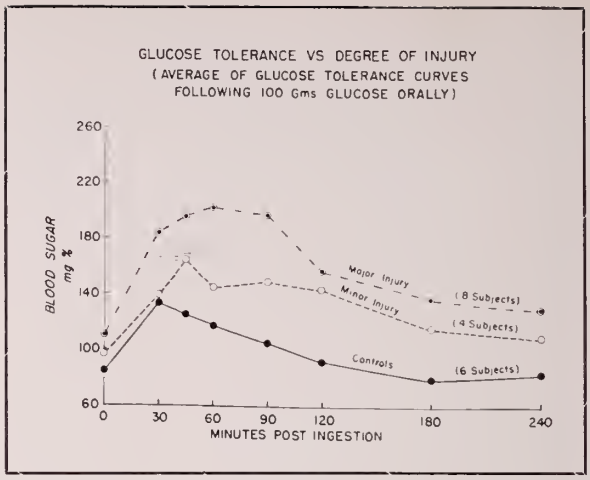


Figure 9. Average glucose tolerance curves in Korean battle casualties with various types of injury. Those casualties with only a minor injury showed an increase in the blood glucose level slightly above controls. Those casualties with major injuries showed a high glucose level that persisted for 90 minutes, followed by a slow fall. It appears that the more severe the injury, the greater the intolerance to glucose. Published with permission of the author[14] and the Annals of Surgery.

Vitamin Metabolism

There is usually a decrease in urinary excretion of thiamine and nicotinamide early after injury. Riboflavin may also be decreased in the urine, but later a brief period of increased excretion of riboflavin is seen. During the convalescent period when the patient is in positive nitrogen balance, there is usually a positive riboflavin balance, when adequate amounts are given. Special emphasis is usually focused on ascorbic acid concentrations. After injury the plasma ascorbic acid concentration diminishes, and its urinary excretion is diminished. The more severe the injury, the greater the disturbance. These changes may continue far into convalescence.[16]

The exact vitamin requirements of the severely injured patient have not been well defined. Since there is evidence of an increased need in vitamins, particularly in burns, the accepted procedure is to give 3 or 4 times the normal requirements of the various components of the vitamin B complex and about 1000 to 1500 mg. of ascorbic acid daily.

Summary

In general, the metabolic response to injury is directly proportional to the magnitude of the trauma. Many of the changes that occur are mediated through the neuroendocrine system. There is an increase in activity of the thyroid and of the adrenal. The 11-oxycorticoids in the urine are increased immediately after injury and may remain increased for several days.

There is diminution in the excretion of sodium and water and in increase in excretion of potassium. Usually there is an expansion of the interstitial space which persists for several days until a diuresis occurs.

One of the most important metabolic changes after injury is the increased outpouring of nitrogen in the urine. This increase in nitrogen excretion may be continued for several days or weeks, depending upon the severity of the injury. As the patient's condition improves, nitrogen losses become less and with adequate intake the nitrogen balance returns toward normal. Later, as the patient's wound heals, there may be considerable storage of nitrogen.

There is a decrease in glucose tolerance after injury, which usually disappears after a few days, unless the injury is massive or there is continuing stimulation.

A rapid loss of body fat occurs during the early postinjury period. These fat losses may continue for a prolonged period. They diminish when nitrogen balance becomes positive. Later in convalescence, if the patient is on adequate intake, an appreciable gain in fat is seen.

These changes in metabolism give an indication of the type of supportive therapy required after injury.

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DISCUSSION

DOCTOR FURMAN, Associate Professor of Research Medicine, University of Oklahoma School of Medicine: Doctor Artz, do you use an anti-catabolic agent such as testosterone? If so, when would you recommend their use, initially, or during the later stages when the big deficit has to be made up?

DOCTOR ARTZ: There is no doubt that these agents enhance the storage of nitrogen as shown by Doctor Abbott's studies. Our own studies showed that they did not continue to enhance the storage of nitrogen over long periods of time and therefore I don't use them. In general, what we try to do is to do as Doctor Abbott has pointed out and try to obtain an adequate intake of nitrogen and calories. Little else is done to disturb the homeostatic mechanisms of the patient other than closing the wound.

DOCTOR ABBOTT: We feel that in most of the patients, forcing food and giving anabolic hormones are probably not necessary. However, I think in the older individual who falls and breaks a hip, very often an injection of testosterone propionate with or without estrogen may be helpful. Certainly, such therapy is often accompanied by a feeling of well-being. In our experience, such patients have shown a better utilization of nitrogen for 12 or 15 days, and we have not felt that it was necessary to do further balance studies because these patients were then usually eating well and over their troubles. In these patients, we have noted a renewed interest in life in many of the old people, and often their caloric intake will also improve. I certainly feel that it is a much better stimulating agent than any of the adrenal steroids. The latter agents are catabolic in effect and the testosterone compounds are therefore preferable to adrenal hormones for such patients.

Clinical Aspects of Magnesium Metabolism

JAMES F. HAMMARSTEN
WILLIAM O. SMITH

KNOWLEDGE OF THE ROLE of magnesium in human physiology is incomplete, but several vital functions of this ion have been established. Many of the intracellular enzymatic reactions in the intermediary metabolism of glucose depend upon magnesium as a co-factor, examples of which are the conversion of glucose-1-phosphate to glucose-6-phosphate and the decarboxylation of pyruvic acid.[1] The extracellular concentration of magnesium may be of considerable importance in muscle function. Magnesium has a sedative action at the neuromuscular junction by a blocking of acetylcholine release, which effect is antagnoized by calcium.[2]

The normal adult human body contains about 20 gms. of magnesium, 98% of which is within soft tissue cells and bone.[3] Striated muscle contains as much as 20 mEq/liter of magnesium. The serum magnesium level is variously reported in the literature from 1.21-2.45 mEq/L. (range of means), depending on the individual laboratories and the method employed. Obviously each laboratory must establish its own normal values for serum magnesium. Spinal fluid magnesium is generally slightly higher than in serum.[4] Gastric juice appears to contain about 1.0 mEq/L. of magnesium.[5] Magnesium concentration in bile approximates that of the blood serum.

The daily requirement for magnesium is about 5 mg./Kg. of body weight. The average-size person can remain in balance on 220 mg. daily.[6] Magnesium is provided in the diet largely by leafy green vegetables, meat, and milk. Absorption from the intestine is probably regulated by the same factors that govern calcium absorption. The great majority of ingested magnesium is ordinarily excreted in the feces; only a small percentage is recoverable in the urine. Renal tubular reabsorption of magnesium occurs to a high degree, probably approaching that of calcium. Whether the tubules can also excrete the cation has not yet been

established, despite several attempts to solve this question.[7,8] As with calcium, there appears to be secretion of magnesium into digestive juices forming a "gut pool," and the small bowel contents seem to be rather rich in magneisum.

The controlling mechanisms for the usual serum cations have been demonstrated, but little is known concerning the regulators of magnesium metabolism. Recent evidence strongly suggests that the parathyroid gland is instrumental in this regard.[9]

A portion of the serum magnesium is bound to serum proteins. This appears to be about 20-30% as compared to 50% for calcium.[10] Evidence is accumulating that there is competition between calcium and magnesium for binding by the protein moieties.[11]

Twenty-six years ago, Kruse, Orent and McCollum showed that rats deprived of dietary magnesium manifested hyper-irritability followed by convulsions and death.[12] Concurrently other workers established that "grass staggers" in cattle was due to magnesium deficiency.[13] Two years later Hirschfelder reported a group of patients with low serum magnesium and symptoms of twitching and convulsions. These patients had renal disease or epilepsy. Several case reports also appeared in the literature linking hypomagnesemia to tetany in patients with severe diarrhea.[15] However, since some observers noted low serum magnesium values in patients without symptoms, symptomatic magnesium deficiency in man was not universally accepted.

Six years ago, Flink and associates reported a well-documented series of patients with delirium tremens who showed low serum concentrations of magnesium.[16] Following intramuscular magnesium sulfate therapy, the symptoms of tremor and hallucinations cleared rapidly with a concurrent rise in serum magnesium. These observers also reported twitching, athetoid movements, and hallucinations associated with a low serum magnesium in patients with massive fluid loss, receiving no magnesium in the replacement solutions. These observations have been amply confirmed.[17,18,19] McCollister, Flink and Doe have recently reported evidence for total body depletion of magnesium in patients with delirium tremens.[20]

The magnesium deficiency syndrome has also been observed in patients with chronic renal disease prior to the development of azotemia,[21] patients with osteolytic disease and hypercalcemia,[22] patients with hypoparathyroidism,[9] and patients undergoing marked diuresis induced by mercurial diuretics.[23] Hypomagnesemia has been reported in some cases of idiopathic epilepsy. The results in a variety of disorders are summarized in figure 1 and table 1.[19]

STATISTICAL ANALYSIS OF SERUM MAGNESIUM IN VARIOUS CLINICAL DISORDERS

	No.	Mean (meq./L.)	S. D.	P
Normals	40	1.48	±0.17	
Delirium Tremens	12	1.14	±0.38	<.001
Portal Cirrhosis	17	1.48	±0.19	
Diabetes Mellitus	39	1.57	±0.13	<.02
Insulin Coma	17	1.70	±0.08	<.001
Renal Disease				
Not Azotemic	11	1.33	±0.36	>.05
Uremia—				
No CNS Depression	12	1.68	±0.35	<.02
Uremia—				
CNS Depression	9	2.49	±0.60	<.001

Table 1

Serum magnesium values in the 12 patients with delirum tremens are shown in figure 1. The mean value is 1.14 ± 0.38 mEq/L., a highly significant decrease as compared to the normals ($P < .001$) (Table 1). However, it may be seen that only five of the group were completely below the normal range and seven were less than 1 S. D. below the normal mean. All of the patients showed marked improvement with magnesium administration.

A patient with ulcerative colitis having severe diarrhea, no oral intake, and intravenous fluid replacement containing no magnesium has been reported previously.[3] The serum magnesium in this case fell to 0.70 mEq/L. and the patient showed the characteristic clinical picture of magnesium deficiency, which improved dramatically with parenteral magnesium administration (Figure 2).

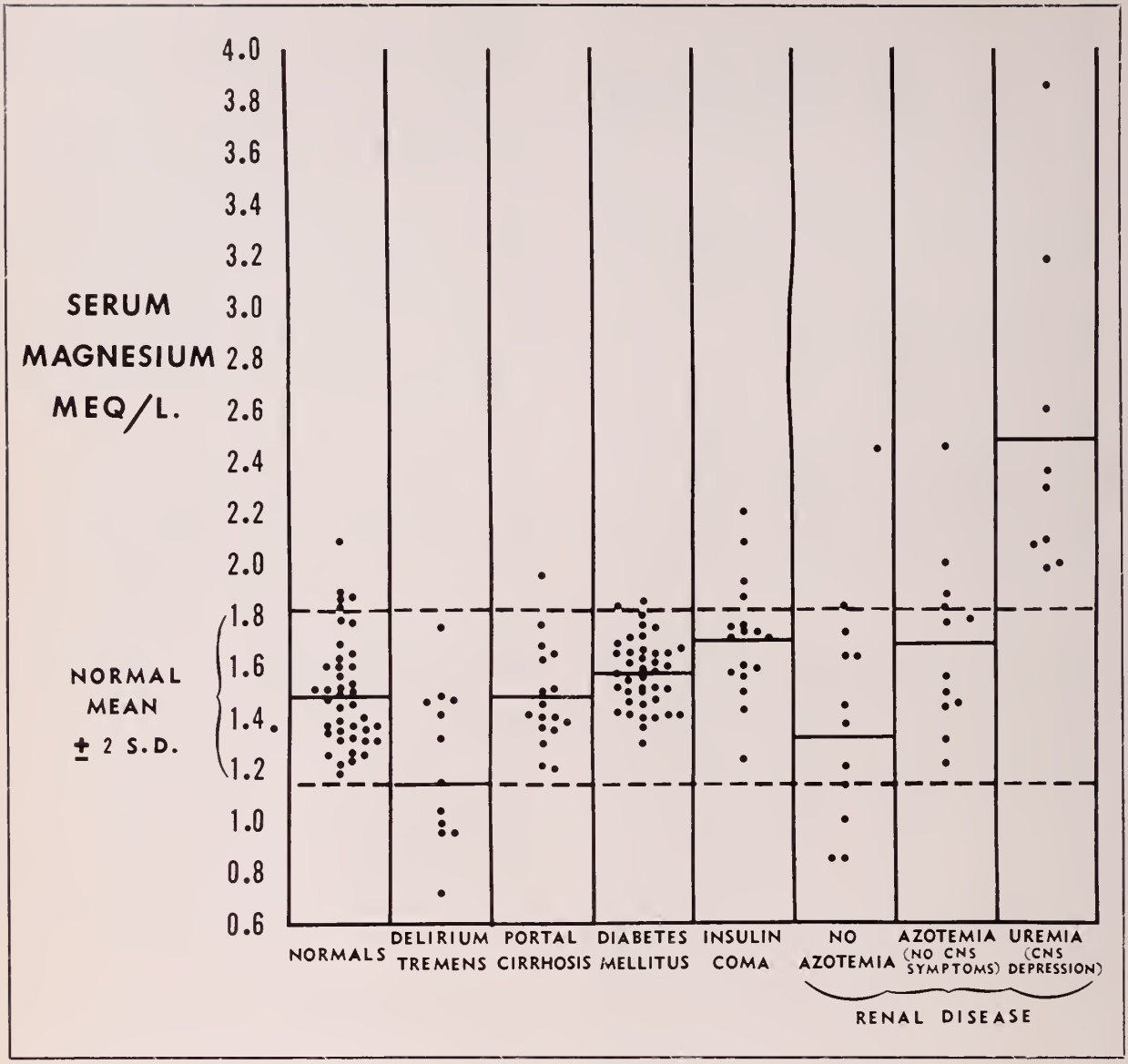


Figure 1. Serum Magnesium in Normals and Various Clinical Disorders.

Treatment of magnesium deficiency at present consists of parenteral replacement with magnesium sulfate. We feel that absorption of magnesium sulfate given orally is inadequate for replacement. The intramuscular route is commonly used and initially 8.0 gms. are given daily in four divided doses. After 48 hours, the dosage may be decreased to 4.0 gms. daily. Obviously, therapy must be given until the deficiency is corrected and maintenance levels must be provided if loss continues. The therapy should not be used in anuric patients and with great caution in oliguria or uremia. We feel it would be wise to add magnesium sulfate to sustaining fluid solutions in patients with deficient oral intake. Daily requirement could be met by 1.0 gm. daily.

Deleterious effects of excessive magnesium have been reported in only one circumstance, uremia. Hirshfelder reported several cases of coma and death in patients with uremia given frequent doses of magnesium sulfate as a cathartic.[14] We have noted high serum magnesium levels in patients with chronic renal disease, nitrogen retention, and central nervous system depression without magnesium administration (Figure 3).[21] Large doses of magnesium in animals pro-

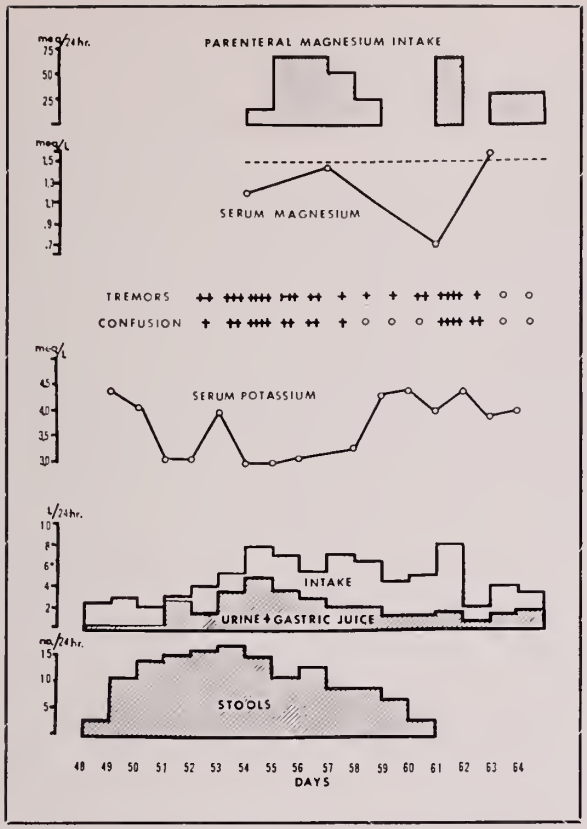


Figure 2.

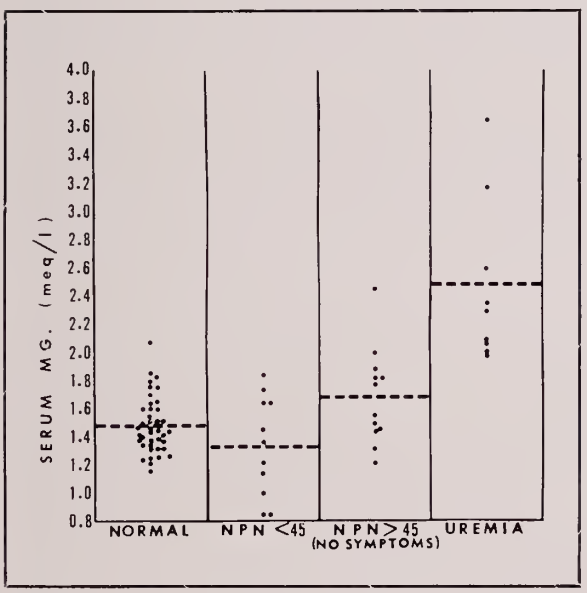


Figure 3. Individual Serum Magnesium Values in Normals and in Patients With Renal Disease.

duces central nervous system depression with coma and death. Our data suggest the possibility that the central nervous system depression and coma in uremic patients may be related to high serum magnesium values. However, some patients with renal disease without azotemia have quite low values and may develop symptoms of magnesium deficiency. The mechanism leading to this hypomagnesemia is not yet established, but appears to be on the basis of an increased urinary loss. Similar findings have also been reported recently in patients with acute renal failure.[24]

Since magnesium is predominately an intracellular ion, we have recently determined the red cell magnesium concentration in normals and patients with delirium tremens and uremia.[25] The values in these groups are given in Table 2. While the evidence is only

	No.	Erythrocyte Red Cell Mg.
Normal	13	5.29± .34 mEq/L.
Delerium Tremens	4	2.30± .70 mEq/L.
Uremia	6	8.91± 1.48 mEq/L.

Table 2.

preliminary, it appears that states associated with a low or high serum value are associated with a corresponding change in the red cell magnesium.

In summary, magnesium is important in biochemical functions of man. A body deficit of magnesium produces a clinical syndrome of mental clouding, confusion, disorientation, hallucinations, tremor, athetoid movements and occasionally actual convulsions. Deep tendon reflexes are generally hyperactive and a cardiac arrhythmia (ventricular irritability) has been noted. This syndrome has not been observed in delirium tremens, massive fluid loss, chronic renal disease prior to the development of azotemia, hypercalcemia, and hypoparathyroidism. Treatment consists of parenteral magnesium replacement. Hypermagnesemia has been observed in patients with uremia, where it is believed to have a role in the central nervous system depression. Artificial dialysis may be indicated when hypermagnesemia is noted in patients with reversible kidney lesions.

It appears that with the development of better methods of determination and an increased awareness on the part of physicians in regard to this cation, magnesium will take its place beside sodium and potassium in clinical medicine.

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DISCUSSION

DOCTOR CONRAD: Doctor Hammarsten, we have two questions for you from one of your fellow panel members.

First, Doctor Relman wants to know, do you think that the increased plasma magnesium in uremia counteracts the neuromuscular effects of the lowered calcium which often occurs?

DOCTOR HAMMARSTEN: I think it is entirely possible. It is well known that there is an antagonism between the two, so that, for instance, it is possible in a normal individual to infuse magnesium and produce hyporeflexia and then correct this by giving the patient calcium. I think Doctor Relman's idea is entirely possible.

DOCTOR CONRAD: And his second question is with reference to one of the slides you showed. He wants to know why the control clearance of magnesium is higher in uremics than in normal individuals.

DOCTOR HAMMARSTEN: The difference was not significant, actually.

DOCTOR RELMAN: Were these subjects with high initial serum magnesium levels?

DOCTOR HAMMARSTEN: They were people who had a slightly high initial magnesium, yes.

DOCTOR CONRAD: In the past, magnesium sulfate has been given to patients with toxemia of pregnancy. Are there any studies of magnesium values in these patients; and secondly, would you consider this therapy contraindicated if these patients were also uremic?

DOCTOR HAMMARSTEN: There have been some studies, but we haven't done any. The studies[1,2] that have been done have shown a normal magnesium. I believe that I would hesitate to use magnesium in patients who were uremic, although I have not had any personal experience with the particular problem. I might mention that the amounts of magnesium sulfates that are used are tremendous compared with what is necessary to replace the usual deficit, and these patients oftentimes get extremely high serum magnesiums.

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Magnesium and Calcium Interrelationships

LEONARD P. ELIEL
WILLIAM O. SMITH
CARL THOMSEN

A. Introduction

MAGNESIUM AND CALCIUM share common chemical and physiologic properties. They are divalent metals of the alkali-earth group, one being subjacent to the other in the periodic table. Their concentrations in body fluids are of critical importance to neuromuscular function, plasma deficits being associated with increased irritability, convulsions and tetany, and plasma excess with decreased irritability, weakness, and paresis. Possible metabolic interrelationships between the two metals have excited sporadic interest for some time. Mendel and Benedict in 1909,[1] and others[2,3] have investigated the effects of oral and perenteral calcium or phosphate loads on the serum concentration and excretion of magnesium in animals. Parenteral injection of calcium in the rat resulted in increased urinary excretion of magnesium[1] while oral and parenteral calcium or phosphate loads in the dog resulted in falls in the blood magnesium levels.[2,3]

In 1933, Bulger and Gausmann reported loss of magnesium (as well as of calcium) in hyperparathyroidism and retention during post-operative recovery[4] (see also references [5,6,7]). Others have reported parallel changes in the urinary calcium and magnesium of patients under androgenic treatment for breast cancer.[8]

These observations suggest that the metabolism of calcium and magnesium are interrelated. We have explored the nature of this interrelationship in osteolytic disease, after calcium and phosphate loads, and after administration of various endocrine and pharmacologic agents.

B. Magnesium Deficit in Osteolytic Disease

Our interest in this field was aroused by the observation of a small group of patients with neoplastic osteolytic disease who developed symptoms of magnesium deficiency in association with hypercalciuria, hypercalmemia, and hypomagnesemia.[9,10] The symptoms were characterized by tremors, athetoid movements, convulsions (in the absence of demonstrable central nervous system metastases, in life or at post-mortem) and, occasionally, by hallucinations, hypertension, and electro-cardiographic disturbances. All manifestations were abolished by the parenteral administration of magnesium sulfate. The data from three of these patients, prior to therapy, are summarized in Table I.

Metabolic studies on these three patients revealed that there was invariably a striking correlation between peaks of urinary

		Diagnosis	Calcium meq/L	Serum Magnesium meq/L	Clinical Findings
1.	M.W.	Carcinoma of breast	8.0	1.0	Clonic convulsions Hypertension Hyperactive reflexes Cardiac arrhythmia
2.	V.A.	Carcinoma of breast	8.5	0.9	Colonic convulsions Hypertension Hyperactive reflexes
3.	C.E.	Multiple myeloma	7.5	1.1	Tremor of Arms Athetoid movements
		Normal	4.5-5.5	1.6± .22 (S.D.)	

TABLE I

calcium and magnesium excretion. An example of these data (from patient 1, Table I) is illustrated in Figure 1.

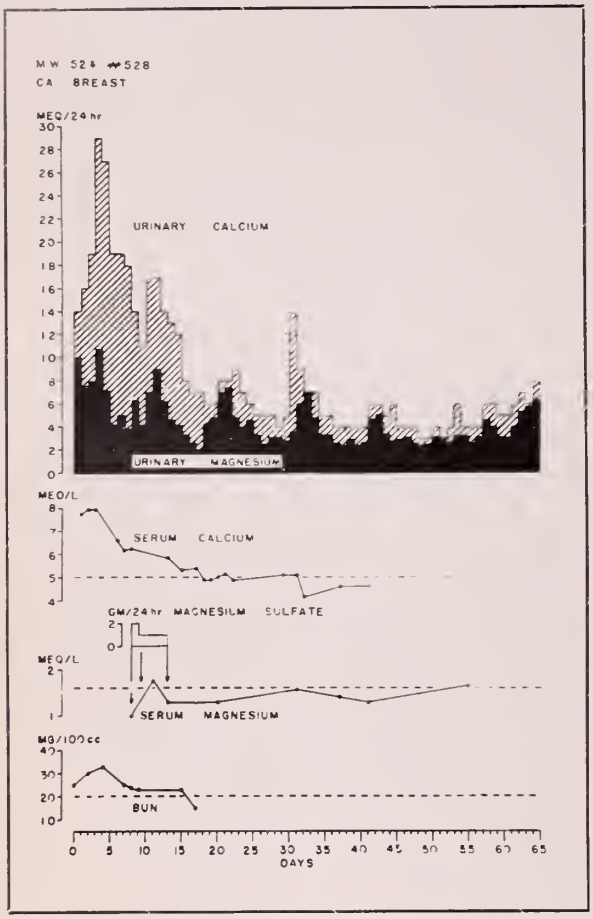


Figure 1. Serum calcium, magnesium, and BUN; and daily urinary calcium and magnesium excretion in case 1 (M.W.), Table I.

Substantially more magnesium appeared in the urine (on reasonably constant magnesium intakes) than would be anticipated on the basis of bone destruction, and the usual ratio of magnesium to calcium in bone which is 1 meq/40 meq.* These data are illustrated in Table II (also from patient 1).

Period	Days	Predicated magnesium loss based on Mg/Ca in bone meq/Period	Actual Mg Loss meq/Period
1	5	-.15	-4.9
2	3	-.07	-5.4
3	7	-.25	-4.2
4	4	-.05	-2.6

TABLE II

*Based on analyses in our laboratory of non-pathological, human cortical bone.

The urinary losses were not accounted for on the basis of extra-cellular fluid depletion.

We assumed, then, that hypercalciuria is associated with urinary magnesium loss and that this leads to an extra-cellular deficit and to depletion of magnesium from another locus which may be intra-cellular or which may represent a preferential withdrawal from bone crystal surfaces (see below). The magnesium depletion, it is further assumed, leads to the production of clinical symptoms.

Since these observations were made, we have examined the nature and pathogenesis of magnesium deficit associated with hypercalciuria and have arrived at a tentative explanation for its occurrence, through the studies now to be described.

1. *Renal Damage.* Renal damage and uremia supervene ultimately in patients with hypercalciuria. Could this be responsible for magnesium loss? While the patients described in Table I had abnormal elevations of the blood urea nitrogen level, we have observed magnesium loss in hypercalciuric patients with normal renal function, determined by the usual clinical criteria. As will be noted below, magnesium loss is also observed in patients with induced hyperparathyroidism and hypercalciuria who have no evidence of renal impairment. We have, therefore, excluded clinically demonstrable renal impairment as a cause of the magnesium deficit.

2. *Hypercalciuria and Hyperphosphaturia.* Patients with destructive bone lesions excrete excessive quantities of calcium and phosphate in the urine. We have, therefore, administered large loads of calcium and phosphate intravenously in an effort to induce magnesium loss.

Intravenous loads of calcium gluconate (up to 50 meq Ca in 4 hours) equivalent to 1000 mg Ca) have been administered to two patients with normal bones and two patients with osteolytic disease (multiple myeloma, and breast cancer) under metabolic balance conditions. All patients had normal renal function. The results in all four cases were similar. Hypercalciuria of 600 mg/day and hypercalcemia of 16 mg% were induced in a typical example (figure 2). The serum

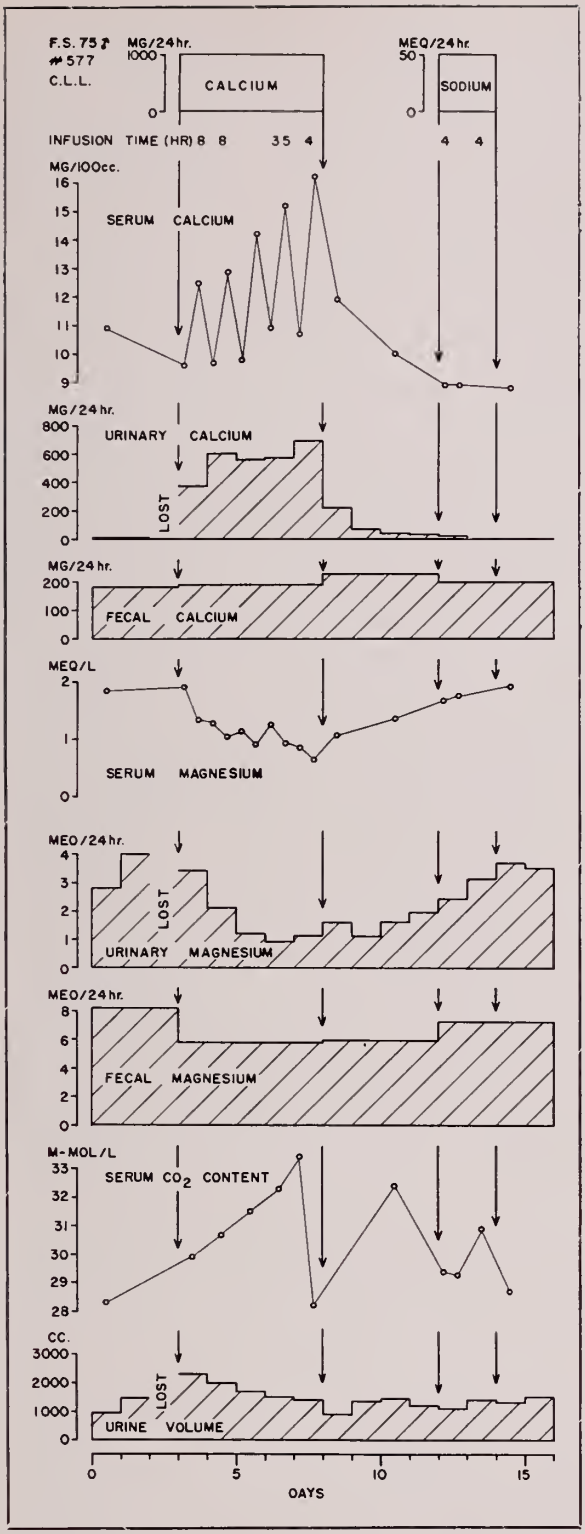


Figure 2. The effects of loads of calcium gluconate and of sodium lactate on the serum concentrations of calcium, magnesium and CO_2; and on the urinary and fecal excretions of calcium and magnesium in a man without osteolytic disease.

magnesium fell from 2.0 meq/L to 0.7 meq/L, but the urine magnesium excretion dropped to 25% of control levels. Infusion of an equivalent amount of cation as sodium lactate failed to reproduce these changes. The balance data revealed addition of magnesium to a body compartment, which we must assume was either bone or intra-cellular, non-osseous tissue. "Intra-cellular" magnesium balance data for a five day loading period, in the example cited, resulted in a retention of 35.2 meq, rather than a loss. We concluded that excess urinary calcium *per se* does not engender magnesium loss.

Intravenous loads of buffered sodium phosphate, providing up to 60 meq of phosphate in a four-hour period, produced no change in the magnesium balance in two patients. As with calcium loading, however, the serum magnesium tended to fall, though not so strikingly (figure 3). We concluded that excess urinary phosphate *per se* does not lead to magnesium loss.

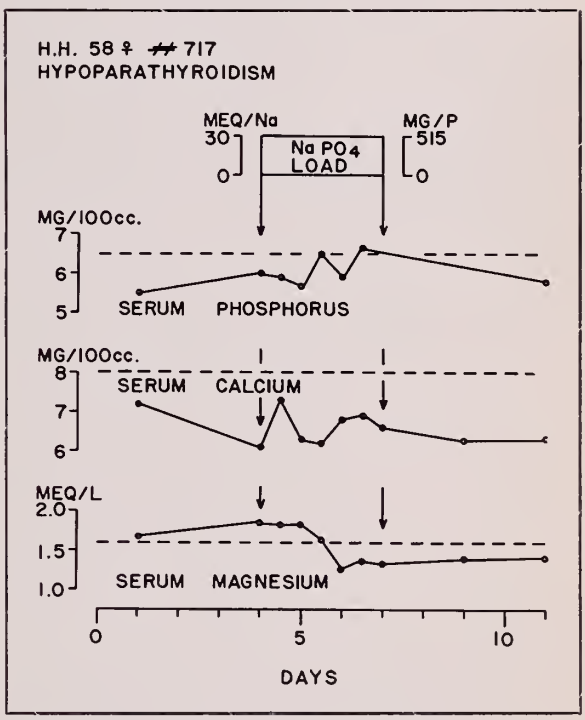


Figure 3. The effect of a buffered sodium phosphate load on the serum concentrations of calcium, magnesium, and phosphorus in a patient with hypoparathyroidism.

3. *Studies in Patients with Hypoparathyroidism.* Patients with hypoparathyroidism afford ideal subjects for the study of wide variations in urine calcium and serum calcium concentrations, in relation to magnesium.

Six patients with hypoparathyroidism (two idiopathic, four post-operative) have been studied. Both cases of idiopathic, and two cases of post-operative, hypoparathyroidism demonstrated serum magnesium levels slightly but significantly below normal (1.2 to 1.3 meq/L—normal mean: 1.6 meq/L), in association with hypocalcemia and hyperphosphatemia.

Administration of parathyroid extract to three patients, maintained on constant diets, resulted in almost exactly parallel changes in urinary calcium and magnesium, from an initial drop over the first two days to subsequent rises above control levels. A typical example is illustrated in figure 4. The serum magnesium, low in two of the patients, likewise paralleled in striking fashion the rise in serum calcium, associated with administration of the extract, and paralleled also the fall after its withdrawal (figure 5).

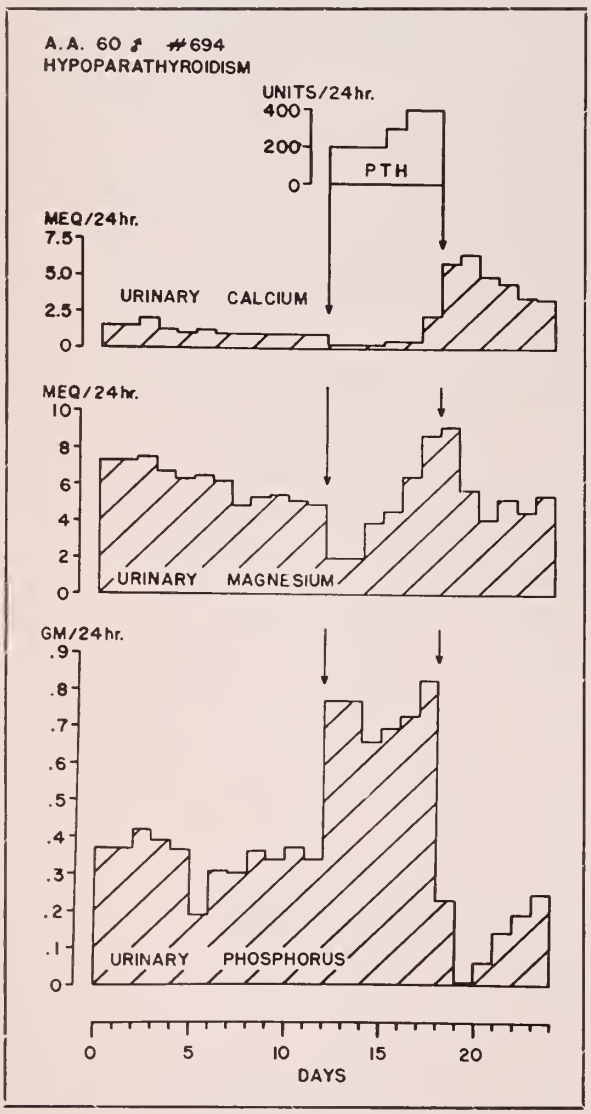


Figure 4. The effects of parathyroid extract on the daily urinary excretion of calcium, magnesium, and phosphorus in a patient with idiopathic hypoparathyroidism on constant dietary intake.

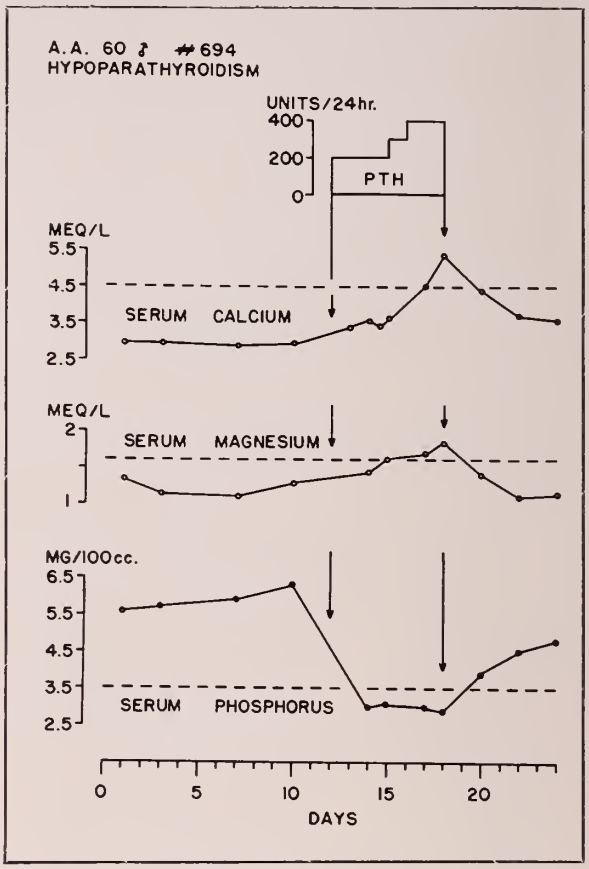


Figure 5. The effect of parathyroid extract on the serum concentrations of calcium, magnesium, and phosphorus in a patient with idiopathic hypoparathyroidism.

Similar observations have been made in one hypoparathyroid patient who received dihydrotachysterol (AT 10) and another who received vitamin D.

It appears from these observations that parathyroid extract and pharmacologically related substances have similar effects on the metabolism of both calcium and magnesium. This is not surprising in view of the close chemical relationship between these two members of the alkali-earth group.

The magnesium balances, obtained from the hypoparathyroid patients receiving parathyroid extract, dihydrotachysterol (AT 10) or vitamin D, were all negative even after correction for losses which might be

attributed to changes in the volume or mass of bone, soft tissue, or extra-cellular fluid. These corrected balances are summarized in the following table:

Patient	Rx	Dose per day	Duration days	Corrected Mg Balance, meq/period
1	Control	—	6	— 0.3
	PTE*	200-400 u	6	—14.2
2	Control	—	5	+ 3.4
	PTE*	400 u	6	—17.3
3	Control	—	5	+ 9.4
	AT 10 ≠	6.25 mg	7	— 8.6
4	Control	—	7	+11.6
	Vit. D	400,000 u	7	—17.5

*—PTE = parathyroid extract

≠—AT 10 = dihydrotachysterol

TABLE III

Thus, administration of parathyroid extract, or large doses of AT 10 or vitamin D, is associated, like malignant osseous disease, with depletion of body magnesium. (None of the patients under investigation had renal impairment.)

Hyperparathyroidism due to parathyroid adenoma appears also to be associated with magnesium deficit. It has been noted by several observers that there is retention of magnesium following removal of the adenoma.[4,5,6,7] Barnes and his co-workers[6] calculated that the retention is greater than that which can be accounted for on the basis of deposition of magnesium in bone, suggesting antecedent deficit. Hyperparathyroidism may be associated preoperatively with extra-cellular deficit (hypomagnesemia);[6,11] or with falling serum magnesium levels after removal of the adenoma,[7] presumably due to repair of the deficit at the expense of extracellular magnesium.

Since administration of substantial doses of parathyroid extract results in tissue magnesium deficit (see Table III), these observations in hyperparathyroidism are expected. It is surprising, though, that serum magnesium concentrations should be low in both hypo- and hyperparathyroidism. While hypomagnesemia in the latter instance seems easily explained on the basis of renal magnesium loss, we believe the former may be ascribed to physical-chemical phenomena (see below).

4. *Intra-cellular Phosphate Depletion.* The large phosphuresis induced by administration of parathyroid extract (see figure 4), and large doses of AT 10 or vitamin D, represents an intra-cellular depletion of phosphate, since it is not accompanied by nitrogen, nor initially by calcium. Is there a requirement for excretion of intra-cellular cation, with phosphate, which is satisfied by magnesium? Actually during the initial phases of therapy only about 10% of the cation required to cover phosphate is met by magnesium, the remainder being made up from sodium, potassium, ammonia and titratable acid.

We have further tested the possibility that intra-cellular phosphate loss requires concomitant magnesium excretion, by the administration, to a patient with hypoparathyroidism, of acetazolamide (Diamox), an agent which induces phosphuresis, in doses of 1000 mg/day for four days. Phosphate excretion doubled, without increase in either nitrogen or calcium excretion, representing, therefore, intra-cellular phosphate depletion. No magnesium loss resulted, however. This experiment has also been confirmed in a normal subject. We have concluded that intra-cellular phosphate depletion does not lead to magnesium deficit.

5. *Citrate Metabolism and Magnesium Deficit.* We were prompted then to seek a factor common to patients with malignant osteolytic disease, and to patients with hyperparathyroid states, which would account for the preferential extraction and excretion of magnesium. An exact parallelism between urinary citrate and calcium excretion was described by Shorr in 1942,[12] a relationship which held whether variations in urine calcium were the result of dietary alterations or of changes in the rate of resorption of calcium from bone. The possibility that citrate could be involved in the production of the magnesium deficit associated with hypercalciuria was rendered more attractive by the demonstration of Hastings, and co-workers, in 1934, that citrate complexes both Ca^{++} and Mg^{++} and that at pH 7 both complexes act as weak electrolytes with dis-

sociation constants of pK's which are practically identical, 3.22.[13]

Under normal conditions of calcium, magnesium and citrate excretion, citrate is theoretically capable of complexing up to ⅔rds of urinary calcium plus magnesium. It is reasonable to postulate that variations in citrate production and/or excretion might result in parallel excretory changes, therefore, in both calcium and magnesium. If citrate binds Mg^{++} as avidly as Ca^{++}, and if magnesium is more readily available for complexing at the surface of the crystal lattice*, then we have a logical explanation for the appearance of more magnesium in the urine than we could anticipate from its very low ratio to calcium in bone. It has already been demonstrated that blood and urine citrate increase following administration of parathyroid extract and vitamin D.[14,15]

Large variations in citrate excretion were induced in a patient with hypoparathyroidism to test this possibility. Hypercalciuria and mild hypercalcemia were first induced with parathyroid extract. During administration of the extract, hydrocortisone was exhibited in doses of 100 mg/day for 6 days and then discontinued,** while the extract was continued for another 4 days. The urinary citrate excretions and corresponding magnesium balances are shown in the following table:

Period	Days	Citrate mg/24 hr	Mg balance meq/24 hr
Control	12	656	+5.0
PTE	8	890	—1.3
PTE + F =	6	288	+0.8
PTE	4	1080	—3.6
Control	4	630	+4.9

TABLE IV

There is an excellent correlation between increases in citrate excretion and the production of a negative magnesium balance, suggesting that changes in citrate production and excretion can account for the development of magnesium deficit.

The hypothesis that increased citrate production in osteolytic disease accounts for the magnesium deficit would make reasonable the assumption that magnesium is lost from bone rather than from soft tissue. Magnesium is firmly bound in soft tissues and is even difficult to extract *in vitro*.[7] The further assumption might be made that the Mg-depleted crystal surface would avidly repair its deficit at the expense of extra-cellular fluid magnesium, given normal citrate production. This appears to be the case in patients with hyperparathyroidism who, following successful removal of an adenoma, manifest symptoms of magnesium deficiency in association with hypomagnesemia and normal serum calcium levels.[6,7]

6. *The Relationship Between the Concentrations of Ca, Mg and P in Serum.* Analysis of all of our data from patients with malignant osteolytic disease, from hypoparathyroid patients, before and after treatment, and from loading studies, reveals that the relationship between the serum concentrations of Ca, Mg, and P is best expressed by an empirical formula $(Ca^{++} + Mg^{++}) \times (P) = 16 \pm 2$ (S.D.) (expressed in milli-equivalents, with an assigned valence of 1.8 to phosphorus). Thus, in the presence of hyperphosphatemia (as in hypoparathyroidism or phospate loading) both Ca^{++} and Mg^{++} tend to be low, while in the presence of hypercalcemia and normal phosprorus concentrations (as in osteolytic disease without renal impairment, or calcium loading), the serum magnesium concentration tends to be low. In hyperparathyroidism with hypercalcemia and hypophosphatemia, we have observed normal serum magnesium concentrations but low values have been reported in patients with parathyroid adenoma.[11] It appears then that the serum magnesium concentration shares in the same physical-chemical mechanism that regulates the calcium and phosphorus concentrations.

C. Vitamin D and Intestinal Absorption of Magnesium

Since vitamin D increases intestinal absorption of calcium, its effects on the absorption of magnesium were tested at a dose

*Magnesium may be held to the surface of the crystal lattice by electrostatic attraction rather than being an integral part of the crystal structure.

**Henneman et al.[16] and Mueller, et al.[17] have shown that cortical steroids depress citrate levels in blood and urine.

=Hydrocortisone (cortisol).

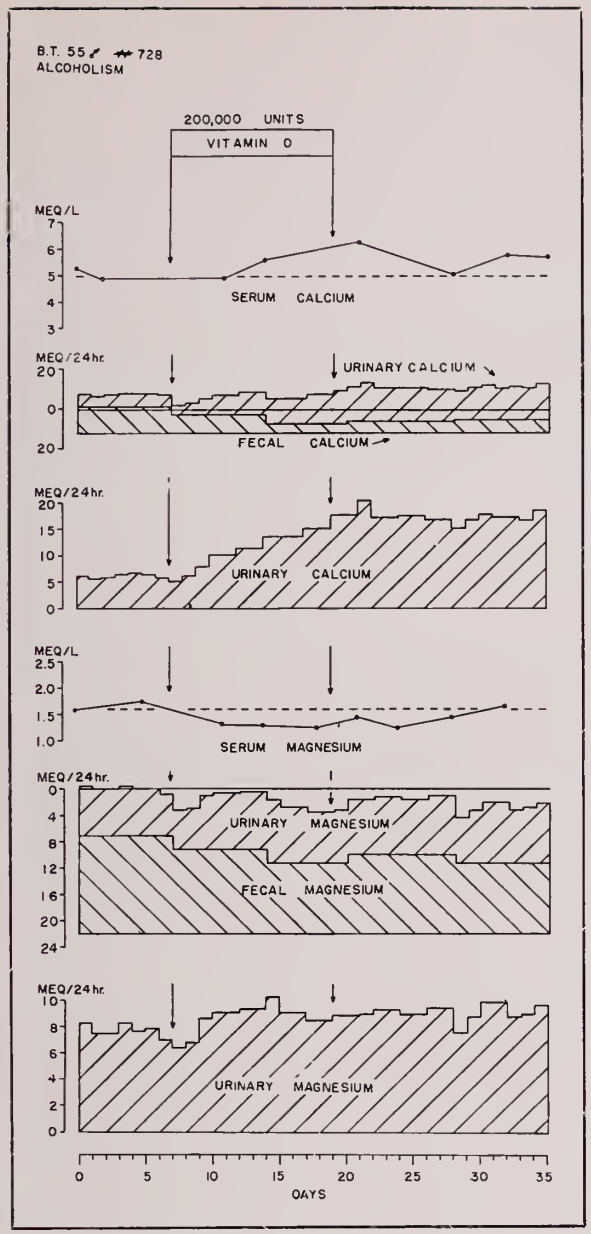


Figure 6. The effect of Vitamin D on the serum concentrations of calcium and magnesium and on the daily urinary excretion of calcium and magnesium in a patient with normal parathyroid function maintained on a constant dietary intake.

The calcium and magnesium balances are plotted in the conventional fashion, the baseline representing intake, a clear space below the zero line representing positive balance, and excretions rising above the zero line, a negative balance.

of 200,000 units per day in a patient with normal parathyroid function. The usual effects on calcium excretion were noted (figure 6), that is, a marked decrease in fecal excretion and a rise in urinary excretion, indicating increased intestinal absorption. Similar, though less marked, changes were noted in the magnesium content of the urine and feces, suggesting that vitamin D does, to a certain degree (at least in the patient studied), increase intestinal absorption of this ion. The balance of magnesium became positive during the period of vitamin administration and remained positive for at least 16 days after it was stopped. The retention of magnesium is greater than can be accounted for on the basis of bone formation and must represent increase in extraosseous tissue magnesium or deposition of magnesium on the bone crystal surface. It is noteworthy that the serum magnesium concentration fell, while that of calcium rose during the period of vitamin ingestion. The storage of magnesium in this patient, in contrast to the loss observed in patient 4 (Table III), may be attributed to the lower dose of vitamin D employed.

D. The Relationship of Serum Magnesium Levels to Tetany

One might suspect that the plasma magnesium concentration could influence the appearance and degree of hypocalcemic tetany. Magnesium sulfate, 5 gm., was infused intravenously into a hypoparathyroid patient (patient 3, Table III), prior to replacement therapy, over a four-hour period, raising the serum magnesium to 3.58 meq/l. During and following the infusion there was virtually complete disapparence of marked Chvostek and Trousseau signs, though the serum calcium concentration remained low. Hammarsten and co-workers (see elsewhere in this symposium) have shown that many patients with uremia exhibit elevated plasma and erythrocyte magnesium levels. This fact may account for the absence of tetany in uremic patients with hypocalcemia resulting from renal failure and hyperphosphatemia. Conversely, one would anticipate that low plasma magnesium levels would facilitate the appearance and augment symptoms of tetany, since convulsive manifestations were observed even in patients with somewhat elevated serum calcium concentrations (Table I).

Summary and Conclusions

1. Destruction of bone due to neoplastic disease may be associated with increased

urinary excretion of calcium, phosphorus, and magnesium; occasionally, hypomagnesemia and symptoms of magnesium deficiency, i.e., tremors, athetoid movements, convulsions, hallucinations, hypertension, and cardiac arrhythmias are observed. Parenteral administration of magnesium sulfate relieves these symptoms promptly. We have attempted to locate the source of magnesium depletion and to study possible factors governing or contributing to the magnesium loss.

2. The loss of magnesium associated with the hypercalciuria is in marked excess of that which one would anticipate from depletion of extra-cellular fluid and loss of bone minerals, indicating either a soft-tissue deficit of magnesium or preferential extraction of magnesium from bone.

3. Impaired renal function, or a renal lesion, can probably be excluded as a cause of the magnesium loss.

4. Exogenous loads of calcium or phosphate, given intravenously, fail to induce urinary magnesium loss but result in falls in the serum magnesium concentration and tissue magnesium storage.

5. The administration of large doses of parathyroid extract, dihydrotachysterol (AT 10), or vitamin D to hypoparathyroid patients all result, like destructive bone disease, in increased urinary excretion of calcium, phosphorus, and magnesium. Balance data reveal that the magnesium losses cannot be accounted for on the basis of changes in the mass or volume of extra-cellular fluid, soft tissues, or of the bone, and indicate a preferential extraction and depletion of magnesium either from soft tissue or bone.

6. Phosphuresis and intra-cellular phosphate depletion are induced by parathyroid extract, dihydrotachysterol and vitamin D. However, intra-cellular phosphate depletion, associated with administration of acetazolamide, does not result in magnesium loss. Available evidence suggests that magnesium is firmly fixed in soft tissues.

7. On theoretical grounds the production and excretion of citrate, which is increased in hypercalciuric states, might account for preferential extraction of magnesium from the bone crystal surface. Preliminary experimental evidence is presented suggesting that this is a reasonable assumption.

8. The concentration of magnesium in serum appears to be governed, in part at least, by the concentrations of calcium and phosphorus.

9. The absorption of magnesium from the intestine may be enhanced by vitamin D, but not in the same degree as calcium.

10. The occurrence of tetany in association with hypocalcemia may be determined in part by the serum magnesium concentration, low levels facilitating, and high levels inhibiting its appearance.

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DISCUSSION

DOCTOR CONRAD: Doctor Eliel, is the formula you presented well enough established so that when you compute the value for phosphorus and calcium, you can rely upon the formula to tell you what the

magnesium concentration should be without actually measuring it?

DOCTOR ELIEL: I don't think the formula is that accurate. Actually, the formula is derived only from the data presented. We haven't tried to apply it to a large enough variety of situations to really establish its validity, but at least it seemed to be an approximate description of the events I presented. I certainly wouldn't want to rely on it for information concerning magnesium levels.

DOCTOR HAMMARSTEN: I would just like to add the comment that really there is no good reason for not measuring the magnesium. It's just as easy to measure as is the determination of calcium, and both can be measured in one determination. This has been the difficulty in this whole field. There have been far too few measurements.

DOCTOR CONRAD: The resemblances between magnesium deficiency states and water intoxication are considerable. Without control studies, and with the doses of magnesium recommended, how can you distinguish between the effect of magnesium itself and any changes in osmolarity it may produce? Will you comment?

DOCTOR ELIEL: As far as our group of patients is concerned, none of them had anything resembling a state of water intoxication. Serum sodiums were normal, for example. We have not done water balances or serum osmolarity determinations which would definitely exclude this possibility.

Clinical Studies in Patients Receiving Long-Term Infusions of Fat Emulsion*

JOHN F. MUELLER
FERNANDO E. VITERI

ALTHOUGH there has been widespread interest in the development of a practical intravenous fat emulsion for human use for many years, the high incidence of untoward reactions to the initial injection has always precluded general acceptance.[1] These acute reactions included: pain, usually in the low-back but often in the chest or extremities; high fever with or without chill; flushing of the skin; nausea and vomiting; dyspnea and cyanosis; rarely, anaphylactoid shock (Table I). However, the accumulated experience of many investigators resulted in an improved fat emulsion which has been available since early in 1956. With its use there has been an almost complete disappearance of untoward reactions with the lone exception of fever.[2] The latter sign occurs in approximately 30% of the patients in whom it is administered, but is mild in nature, delayed, short-lived and in the great majority of patients clinically non-significant. Therefore, from the standpoint of clinical acceptiveness, a usable product is available.

The second problem then had to be faced, i.e., the effect of repeated infusions of fat in the same individual. Until recently there was very little information available concerning this problem in patients receiving the improved emulsion. It would seem that one of the major indications for such a product would be its use in patients requiring long-term therapy. In the Spring of 1957 at the meeting of the Surgeon General's Task Force on Intravenous Fat Emulsions, Upjohn and Levinson[3] and Watkins[4] reported independently on the development and nature of a severe reaction in three volunteers who received multiple infusions of a fat emulsion. This syndrome was characterized by high fever, hepatosplenomegaly, jaundice, anemia, bleeding tendency, abdominal pain, nausea and vomiting. Fortunately all the subjects survived without recognizable sequelae. Although a large battery of laboratory studies were performed, it was not possible in these individuals to establish the exact patho-physiology of the syndrome, but since the recipients of the fat emulsion were ingesting a normal diet it was suggested that this might be an "overloading" syndrome from excess fat. This hypothesis seemed to have some credence since the syndrome had not been observed before in sick patients infused with fat emulsions and receiving most of their caloric demand by vein. Furthermore, there was a suggestion, unproved, that persistent lipemia accompanied the syndrome.

It was clear, therefore, that further observations were necessary in patients receiving multiple infusions of a fat emulsion. An attempt has been made and is still in progress to study systematically various clinical and laboratory parameters in such patients. This report concerns those studies which have been completed. Two patients

*This study was performed under a grant from the Research and Development Division, Office of the Surgeon General, United States Army (Contract No. DA 49-007-MD-800).

CHART DEPICTING REACTION RATES USING THE "OLD" AND "NEW" FAT EMULSION FOR INTRAVENOUS ADMINISTRATION ACCORDING TO NUMBER OF SUBJECTS AND NUMBER OF INFUSIONS

Emulsion	OLD (Aug. '54—Dec. '55)*				NEW (Jan. '56—Oct. '56)			
Number of Infusions:	293				362			
Number of Subjects:	110				151			
Reactions	Number of Infusions	% per Infusion	Number of Subjects	% per Subject	Number of Infusions	% per Infusion	Number of Subjects	% per Subject
Fever**	45	15	32	29	60	17	45	29
Chill	21	7	18	16	2	.6	17	1.3
Back and Chest Pain	38	13	33	30	0	0	0	0
Flushing	7	2	7	6	1	.3	1	.7
Nausea and Vomiting	15	5	14	13	4	1	4	2.6
B.P. Rise and Fall	20	7	14	13	0	0	0	0
Dyspnea Cyanosis	14	5	14	13	0	0	0	0
Shock	2	0.7	2	2	0	0	0	0
Rash	0	0	0	0	1	.3	1	.3
Miscellaneous (Twitching, Itching, Hives, Abd. Cramps)	6	2	6	5	3	.8	3	2
Overall				51%				

*The author is indebted to Doctor Hugo Moeller and Doctor Morton Grossman for some of the data on the "old" emulsion used in this report.

**Over 100.6 orally or 101.6 rectally.

TABLE I

have been investigated intensively and purposefully, while in four others, it has been possible to make only spotty observations.

Methods

M.J. was a 45 year old white woman admitted to the Medical Service of the Cincinnati General Hospital because of malnutrition. Complete physical and laboratory examination failed to reveal any pathologic cause. She weighed 77 pounds at the time of admission. Six hundred cc. of Lipomul,* intravenous, was administered daily for 17 days.

E.B. was a 44 year old white woman admitted to the Psychosomatic Ward of the Cincinnati General Hospital because of a long history of functional gastrointestinal symptoms, severe psychoneurosis, hysterial syncope, weight loss and diarrhea. She weighed 83 pounds at the time of admission. After intensive diagnostic work-up failed to reveal any significant organic pathology, she was given 600 cc. of Lipomul* intraenously each day for 24 days.

Both subjects were given the Lipomul early in the morning according to the technique previously described.[2] They were examined each day by a physician for any change in the physical status. Laboratory procedures were carried out at appropriate intervals.

The routine laboratory tests were performed according to standard techniques. The TAMe assays for prothrombin were carried out in Doctor Helen Glueck's laboratory according to the procedure previously published.[5] The optical density of the serum was measured in a Coleman Jr. spectrophotometer at 700 *mu* using water as the blank. Cr^{51} and Fe^{59} were used to study life span and iron turnover and red cell uptake respectively according to established methods, with the help of Doctor Ben Friedman of the Radio-isotope Laboratory. In this laboratory, the apparent red cell half-life is normally 30 days. Plasma iron turnover is 0.4 - 0.8 mg/day/100 ml blood. Red cell uptake is normally over 75% after 14 days.

Results

1. Clinical: In both patients the infusions were discontinued at 24 and 17 days respectively because of the occurrence of a clinical picture which was similar in nature and vomiting (Figures 1 and 2). In both instances there was some decrease in oral caloric intake prior to the onset of the re-

*Lipomul, Upjohn, is a 15% cottonseed oil emulsion, emulsified with 1.2 Gm.% soybean phosphatide, .3% Pluronic F68, 4% Dextrose and was kindly furnished by Doctor E. A. Hawk, The Upjohn Company, Kalamazoo, Michigan.

but more severe in E.B. than M.J. This syndrome was manifested by fever, hepatomegaly, abdominal pain, anorexia, nausea

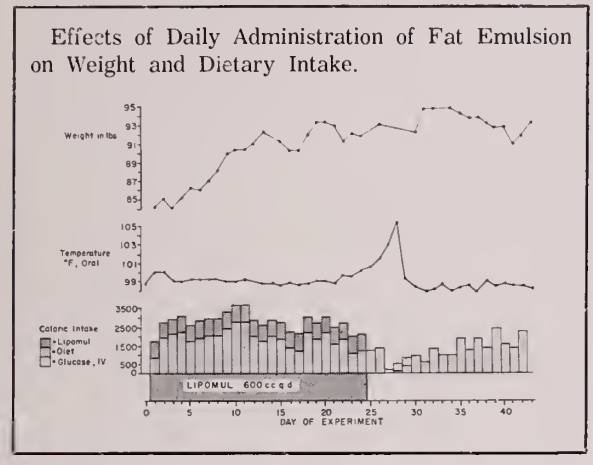


Figure 1.

action. In E.B. intravenous hydrocortisone produced an immediate lysis of the fever and an uneventful recovery ensued. Hepatomegaly subsided over the period of several days. In the case of M.J. simple discontinuation of the fat emulsion resulted in a gradual return of her pre-infusion state of health. In neither instance were any demonstrable residuals noted.

It should be noted that a significant weight gain occurred in each patient. There was an associated increase in appetite early in the infusion period as evidenced in Figures 1 and 2. Both patients related subjecrise in reticulocytes. At the height of the anemia, fecal urobilinogen was normal and occult blood was not found in the stool. No appreciable change in the white cells or

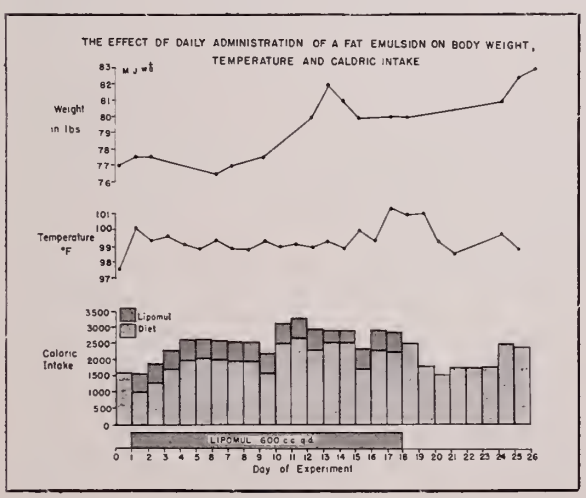


Figure 2.

tive increase in sense of well-being during the early infusion period.

2. Hemogram: In E.B. there was a slow decline in hematocrit, red cell count and hemoglobin after the second week (Figure 3). This was associated with a slight

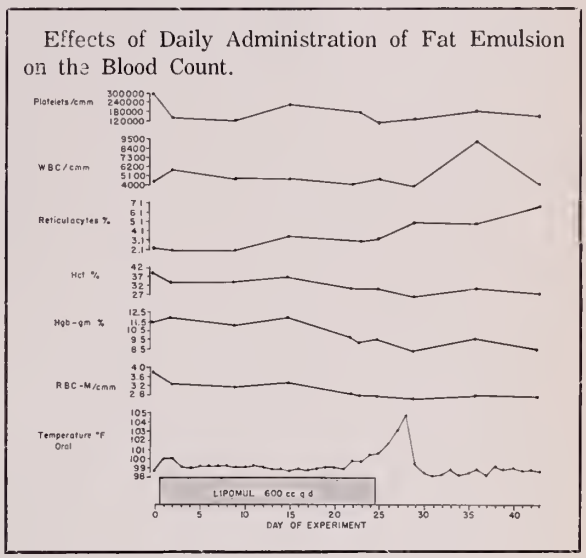


Figure 3.

platelets was found. The isotope studies were performed on the first day and repeated on the day following the acute febrile illness. The apparent red cell half-life was 17.7 days on the first study and 14.3 days on the second. Plasma iron turnover was 0.6 mg/day/100 ml of blood initially and 0.44 mg/day/100 ml at the end. The red cell uptake of radio-iron after 14 days was normal in both instances. The serum iron levels were low normal and likewise did not change. It should be mentioned that a total of 545 cc. of blood were withdrawn from this patient over the period of the study.

The hemoglobin in M.J. showed a slight fall after the first week, but no progressive change throughout the remainder of the experiment (Figure 4). An anemia developing in the post-infusion period has not been explained. No significant change was apparent in the white cells or platelets. No isotope studies were performed in this subject.

Bone marrow aspiration in E.B. before and after the experimental period showed slightly more fat in the post-infusion specimen but otherwise no significant change was evident.

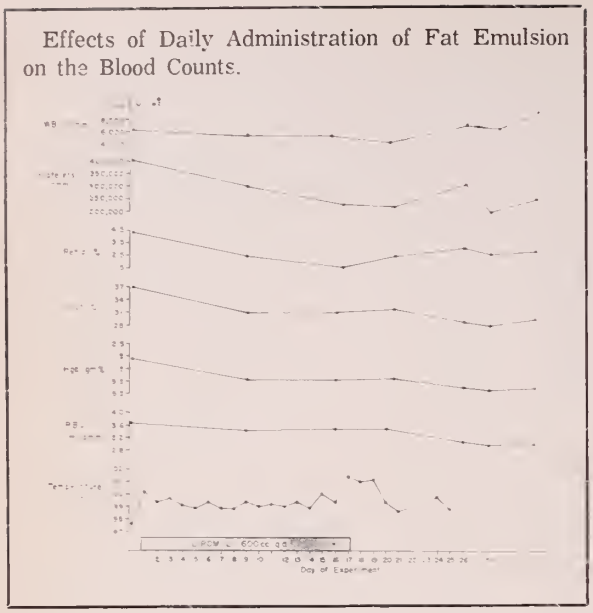


Figure 4.

3. Liver Studies: Complete function studies were performed on E.B. The only abnormality was a progressive rise in BSP retention (Figure 5). This peaked at 23.5% the day following the lysis of fever and then promptly returned to normal. In M.J. only the BSP and bilirubin were measured. The former showed a rise from 1.5% to 10%, but no change in bilirubin occurred.

In M.J. a liver biopsy was obtained before and 5 days after the infusion period. At the time of the second biopsy, the liver

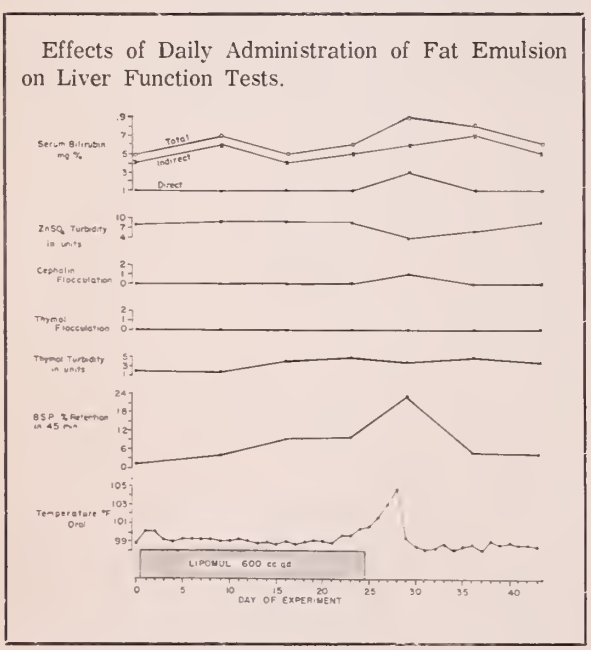


Figure 5.

was still grossly enlarged, although somewhat smaller than at the height of the reaction. The initial biopsy was reported as showing no diagnostic abnormalities. The second biopsy revealed scattered microgranulomas of non-specific nature. There was no evidence of pigment or abnormal lipid deposition. Repeat biopsy 10 weeks after the second showed one area of non-specific focal necrosis and an otherwise normal histologic picture.

4. Lipid Studies: The serum remained clear twenty-four hours after each infusion in both cases as measured by optical density. Clearing studies likewise did not show any tendency toward slower clearing with successive infusions. However, there was a slight rise in total fatty acids, presumably soluble lipoprotein, which in E.B. was almost exclusively due to a large increment

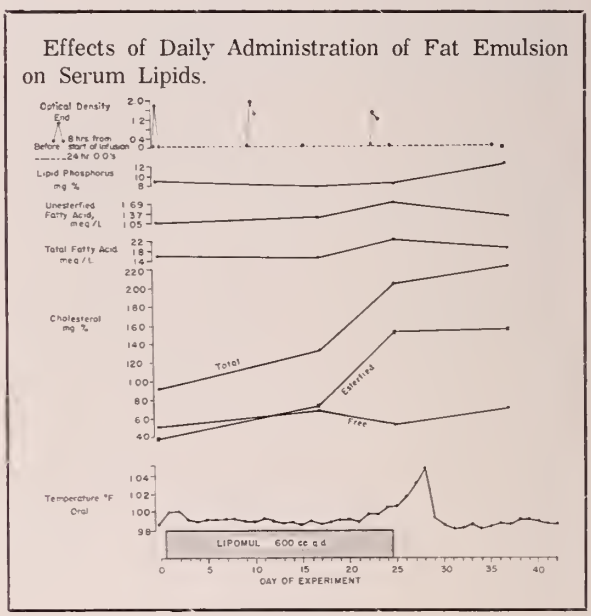


Figure 6.

in serum esterfied cholesterol (Figure 6). Only a slight rise in the serum cholesterol occurred in M.J. (Figure 7).

5. Coagulation Studies: In both patients, studies were calculated to assay not only the long-term effect of multiple infusions of fat, but also the immediate effects of fat infusion on the coagulation mechanism. The latter would appear to reflect hypercoagualability at the height of lipemia as evidenced by decreased silicon clotting times and increased prothrombin conversion.

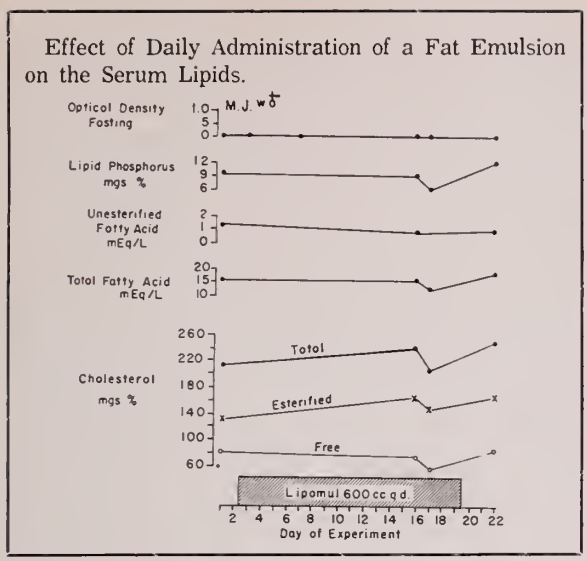


Figure 7.

However, progressive fall in the plasma prothrombin and slight falls in the accessory factors after many infusions reflect hypocoagulability (Tables II and III). The oral administration of 10 mg. of Vitamin K_1 oxide daily to M.J. had little effect upon the slightly lowered prothrombin time, raising it from 40.2 TAMe units to 44.1 units in 72 hours.

Discussion

There seems to be slight doubt that both of the subjects experienced a reaction to the long-term administration of an intravenous fat emulsion. The mildness of the reaction in M.J. may reflect the fact that she received less in total amount, or more likely that there are and will be individual variations in susceptibility to the reaction. The syndrome was strikingly similar to those previously reported.[3,4] However, until more cases have been studied, its expected incidence is impossible to predict. The studies performed in these two patients may give some hints as to possible etiologic factors.

Both of the reported patients were consuming an adequate diet during the infusion period. This was also true of the previously described cases. This suggests that the excess fat in the emulsion, which presumably is not required, may be inefficiently handled, back-up in the tissue and finally in the blood and thereby produce the "overloading" syndrome. The lipid studies in this report do not support the existence of lipemia but do not eliminate the possibility of tissue deposition. The liver biopsy on M.J. at the time of reaction, however, did not show any fatty infiltration but rather some non-specific micro-granulomas. The interpretation of the significance of the latter is open to question.

The cause of the anemia in E.B. is not explained. Unfortunately the initial red cell half-life was abnormal so that the slight shortening from 17.7 to 14.3 days is difficult to interpret. However, the normal serum bilirubin and fecal urobilinogen would seem to rule out a hemolytic process. The

EFFECTS OF INTRAVENOUS FAT ON COAGULATION MECHANISMS

Date	7-31-57	8-6-57		8-12-57		8-20-57		8-23-57		9-3-57	9-6-57	9-17-57
		Before	After	Before	After	Before	After	Before	After			
Siliconized Lee White Clotting Time												
1st Tube	4 min.	14 min.	6min.	9 min.	——	——	6min.	9 min.	6 min.	22 min.	8min.	6min.
2nd Tube	——	19 min.	8min.	14 min.	——	——	12min.	14min.	——	41 min.	12min.	12min.
3rd Tube	26min.	25min.	——	20min.	——	——	12min.	18min.	——	60min.	16min.	17min.
Plasma Prothrombin Time												
Control	11.4 sec.	——	13 sec.	13.0sec.	——	——	13.0sec.	12.7 sec.	——	11.6sec.	11.1sec.	10.5sec.
Patient	11.9 sec.	——	11.5sec.	11.5 sec.	——	——	12.5sec.	11.2sec.	12.6sec.	16.0sec.	13.7sec.	11.2sec.
	(95%		(100%	(100%			(100%	(100%	(100%	(57%	(70%	(97%
Serum Prothrombin Time												
28° Serum												
20 min. inc.	11.3 sec.	——	<60sec.	8.8sec.	——	——	8.5sec.	8.7sec.	10.9sec.	10.5sec.	10.2sec.	9.5sec.
40 min. inc.	132 sec.	——	<60sec.	9.9 sec.	——	——	9.5sec.	11.0sec.	10.9sec.	11.9sec.	12.1 sec.	9.8sec.
60 min. inc.	602sec.	——	<60sec.	22.4sec.	——	——	17 sec.	13.6sec.	48.8sec.	11.6sec.	13.4sec.	15.2sec.
Plasma Tame Assays	35u/ml	——	38 u/ml	32 u/ml	——	——	28u/ml	32 u/ml	39 u/ml	10 u/ml	28 u/ml	49 u/ml
Serum Tame Assays												
20 min.	27u/ml	——	1 u/ml	20 u/ml	——	——	5.0u/ml	34 u/ml	7 u/ml	4 u/ml	0	36 u/ml
40min	12 u/ml	——	1 u/ml	11 u/ml	——	——	5.0 u/ml	5 u/ml	7 u/ml	0	0	13 u/ml
60min.	8 u/ml	——	2 u/ml	6 u/ml	——	——	8.2 u/ml	6 u/ml	6 u/ml	0	0	7 u/ml
Factor V	normal		normal	normal			normal	normal	normal	slight fall	normal	normal
Factor VII	normal		normal	normal			normal	normal	normal	normal	normal	normal

TABLE II.

moderate depression of plasma radio-iron turnover suggests decreased erythropoiesis, but normal red cell radio-iron uptake and mild reticulocytosis make this possibility less likely. Further studies are certainly indicated and are in progress. It would seem, however, that anemia is not a prominent etiologic factor in the febrile syndrome.

Hepatic necrosis or failure is not suggested by the findings. The elevation of the BSP has been a common observation by most investigators. It is thought that this reflects merely a temporary "clogging" of the Kupffer cells of the liver by fat or its products and thus reduces their efficiency in clearing the blood of the injected dye. Certainly the abnormality rapidly disappears following cessation of the infusions. No other abnormalities are apparent in the liver profile which support previous data on the effect of I. V. fat emulsions in patients with liver disease.[6] The non-specific microscopic abnormalities in the liver reported in M.J. are not entirely explicable but would not appear to be extensive enough to be considered causal of the reaction. Unpublished observations by our group have failed to reveal any abnormality of the fasting serum proteins by electrophoresis in patients receiving multiple infusions of Lipomul.

The coagulation studies are considered important although their relationship to the chronic reaction is not clear. Phospholipids have known thromboplastic activity. Their presence in the emulsion as emulsifiers may well be responsible for the hypercoagulability noted immediately following each infusion. These observations have been extended in Doctor Glueck's laboratory and show that in most patients there is a shortening of silicon clotting times and increased prothrombin conversion at the end of the fat infusion. The particulars of these studies will be the subject of another report. The hypocoagulability noted at the end of the infusion period in E.B., and suggested by the bleeding tendency in previously re-

THE EFFECT OF INTRAVENOUS INFUSION OF A FAT EMULSION ON BLOOD COAGULATION

	10-15-57		10-16-57		10-17-57		10-22-57		10-29-57		11-5-57	
M.J. wŏ	Before	After	Before	After	Before	After	Before	After	Before	After	Before	After
Siliconized Lee-White Clotting Time (min.)												
1st. Tube	12		12		16	4	12	5				
2nd. Tube	19		22		24	8	14	7				
3rd. Tube	23		30		25	16	17	8				
Plasma Prothrombin Time (min.)												
Control							12.4		11.2		10.6	
Patient	14				10.4	11.4	11.3	12.7	11.3		10.3	
Serum Prothrombin Time (min.)												
20 min.	12.3		8.9		10.3	9.7	9.8	10.5				
40 "	14.5		10.2		37.5	14.2	21.6	22.7				
60 "	19.2		11.2		29.2	22.5	43.5	>60				
Plasma Tame Assay (units)	55.5		61.6				36.6	32.4	40.2		41.0	
Serum Tame Assay (units)												
20 min.	9.3		24.5		1.2	3.7	25.9	2.8				
40 "	5.4		14.3		1.9	7.3	1.7	0				
60 "	0		11.6		1.7	3.6	3.1	3.0				

TABLE III.

ported cases, might be explained by repeated episodes of hypercoagulability at the height of lipemia following each fat infusion with eventual depletion of certain clotting factors. This is strictly conjectural and will require much more proof before being accepted as fact. However, the effect of fat on coagulation mechanism is of vast importance not only as concerns the problem of intravenous feeding, but its role in the causation of coronary thrombosis. Further careful work is needed in this field.

The prompt, almost dramatic, response of the febrile reaction to the intravenous hydrocortisone in E.B. may be a hint as to the nature of the reaction. The clinical observation was striking. It suggested that hypersensitivity might be invoked. The microgranulomas of the liver would be consistent with such a concept. It would be difficult to prove or disprove such a hypothesis with the data now available.

Therefore, one must conclude that at the moment there is no good explanation for the febrile reaction to multiple infusions of fat emulsions. It may be a consequence inherent in the administration of so much lipid to an individual in positive caloric balance. This is suggested by the fact that multiple infusions to sick patients, all of whom were calorically deficient, both by the author and others, has not resulted in such a severe clinical state as described above. (The similarity of the clinical state to that of idiopathic hyperlipemia is striking and allows for much idle speculation in that direction.) However, only much more experience will answer this important question. At the moment it would seem that prolonged administration of a fat emulsion to any patient should be accomplished with care and the realization that such a reaction as herein described might occur.

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Physiological Observations on Fat Transport Utilizing an Intravenous Emulsion*

JOHN F. MUELLER
JACK IACONO

IN A PREVIOUS PUBLICATION, the relationship between the acute febrile reaction to a single infusion of a fat emulsion and the rapidity of its removal from the blood stream was discussed.[1] That study grew out of the observation that purification of the soybean phosphatide emulsifier reduced drastically the incidence of acute reactions to a fat emulsion with one prominent exception—fever. The latter occurred in exactly the same percentage of patients when the improved emulsion was given as it did when the highly reactive emulsion was administered. More recent confirmatory data accumulated in 31 patients receiving 127 infusions at the Cincinnati General Hospital shows that 26% of the subjects had a delayed type febrile reaction. Thus it has been suggested that the febrile reaction is perhaps "physiologic" in nature. This is supported by the fact that rapid disappearance of the infused fat from the blood is associated with a high incidence of febrile reactions whereas delayed clearance is accompanied by a very low incidence of fever (Figure 1). It has been ventured that endogenous heat produced by rapid oxidation of the rapidly cleared fat in a susceptible individual might be responsible for the fever. However, there was no proof that rapidly cleared fat was necessarily rapidly oxidized. Therefore, an attempt to correlate ketone production with fat clearance was made and is the basis of part of this report.

The marked variation of clearing patterns with the same individual under variable circumstances as well as between different individuals was pointed out (Figure 2). This suggests that there is a highly significant physiologic mechanism for clearing infused fat from the blood and that this mechanism is probably effected by various stimuli.

*This study was performed under a grant from the Research and Development Division, Office of the Surgeon General, United States Army (Contract No. DA 49-007-MD-800).

Relation Between The Optical Density of the Plasma 8 Hours After The Start of an Infusion of 600 ml of Lipomul and the Highest Rectal Temperature in the 12 Hour Post-Infusion Period in 95 Subjects Receiving Their First Infusion.

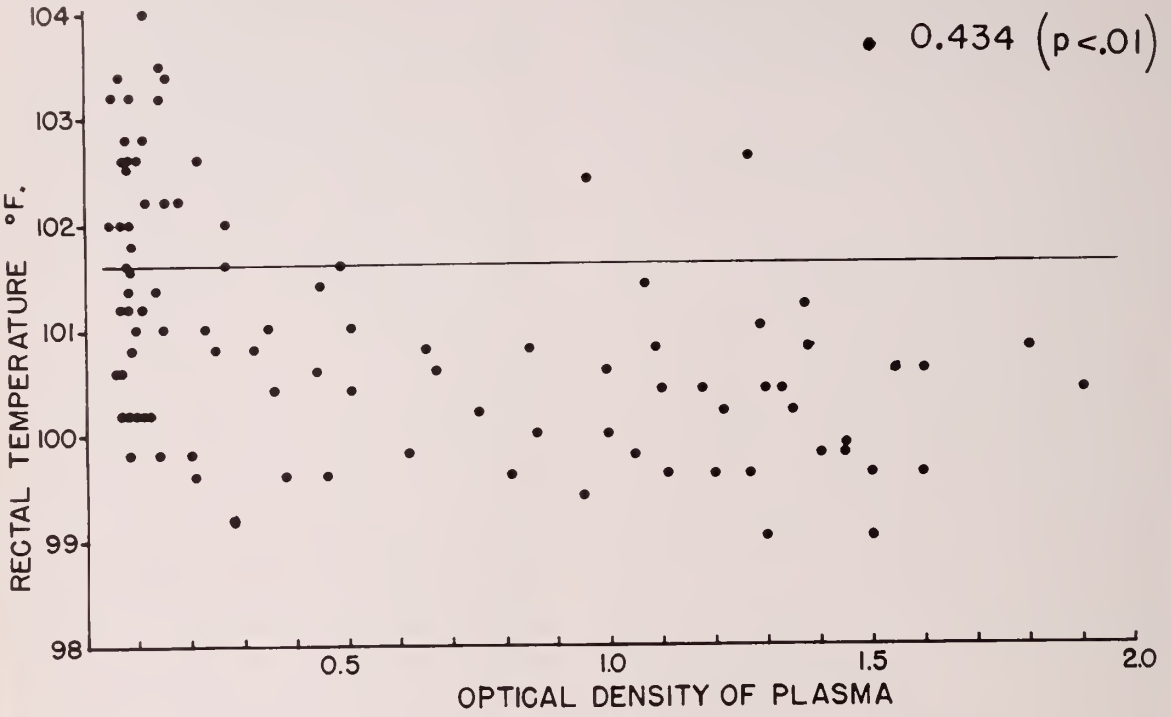


Figure 1.

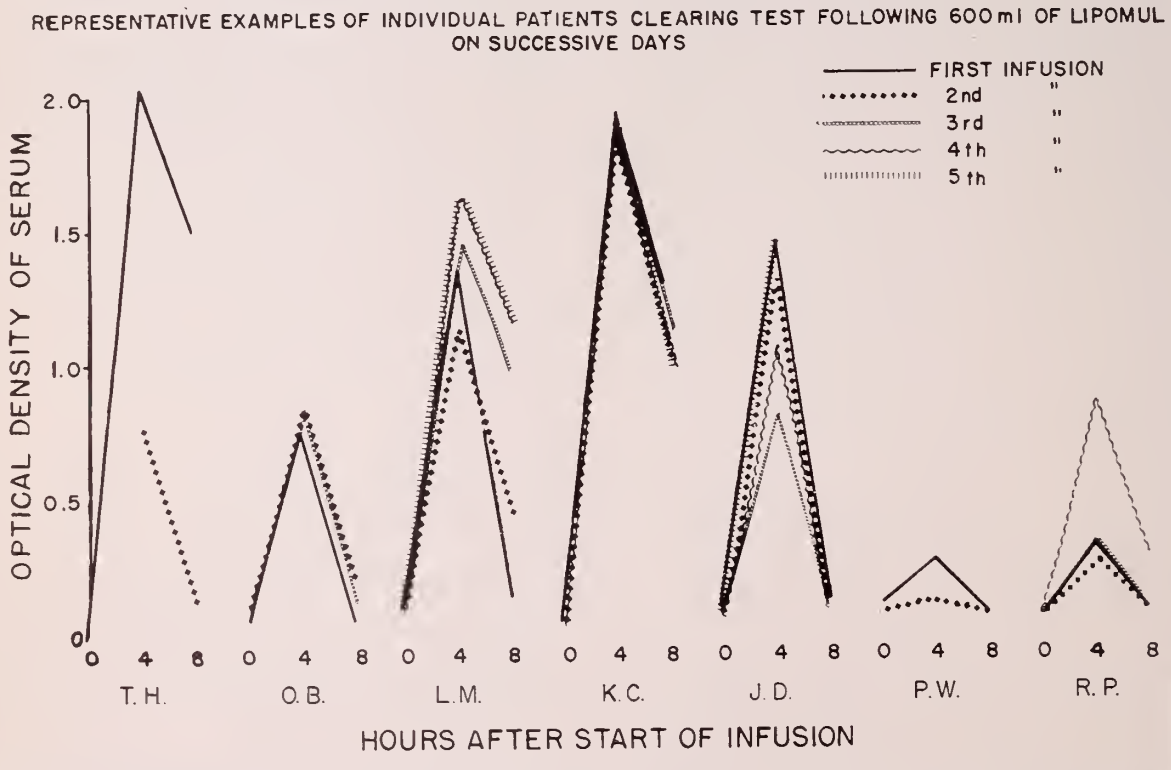


Figure 2.

In an effort to better understand the physiologic process behind clearing of the blood, "clearing factor enzyme" or lipoprotein lipase was studied in our patients. This is the substance first described by Hahn which can be demonstrated in the blood of animals and man following the administration of heparin or heparin-like substances, and which produces clearing by hydrolysis of the fatty acid moiety. Iacono and Cleland from our laboratory showed that without heparin, the concentration of this enzyme was so low as to account for only 5% of the clearing of the infused fat emulsion.[2] Thus if lipoprotein lipase is physiologically significant in the actual clearing of the blood, it must function at the cell level rather than intravascularly.

In an attempt to gain some insight into the perplexing problems, various clinical parameters and disease entities were correlated with clearing patterns without avail (Table I). At this point it was decided to look at the role of the various serum lipids during clearing. The first study was performed in nine hospitalized patients and nine healthy volunteers. These preliminary results led us to intensively study the changes in serum lipid patterns under varying conditions following intravenous fat in 10 healthy volunteers. These findings corraborated the preliminary ones and are the basis of the latter half of this report.

Methods

For the ketone study, 11 patients from the wards of the Cincinnati General Hospital were randomly chosen and infused with 600 cc. of Lipomul. The patients were fasting during the entire procedure. The Lipomul was administered at a constant rate of 64 ggts/min., requiring approximately 3½ hours total time. Blood was drawn before, at the end, and 8 hours after the beginning of each infusion and analyzed for total ke-

THE RELATION OF VARIOUS PHYSIOLOGIC PARAMETERS AND DISEASED STATES AND THE PATTERN OF CLEARING OF THE BLOOD FOLLOWING AN INFUSION OF A FAT EMULSION

		Clearing Pattern				
	Total	Intermediate*		Slow**		Fast***	
Weight	No.	No.	(%)	No.	(%)	No.	(%)
Less than 100 lbs.	8	6	(75)	2	(25)	0	(0)
100 - 150 lbs.	76	27	(35)	28	(36)	21	(29)
150 - 200 lbs.	14	6	(43)	6	(43)	2	(14)
Over 200 lbs.	1	0	(0)	1	(100)	0	(0)
Blood Pressure							
Diastolic above 90 mm Hg.	28	16	(57)	8	(28)	4	(15)
Diastolic below 90 mm Hg.	77	28	(36)	29	(37)	20	(27)
Age							
20 - 40 years	21	7	(33)	12	(57)	2	(11)
40 - 60 years	39	11	(28)	20	(51)	8	(21)
60 - 80 years	54	28	(52)	11	(20)	15	(28)
Clinical Diagnosis							
Hyperlipemia	1	0	(0)	1	(100)	0	(0)
Diabetes Mellitus	2	1	(50)	1	(50)	0	(0)
Coronary occlusion	5	1	(20)	4	(80)	0	(0)
Stroke	7	3	(43)	4	(57)	0	(0)
Arteriosclerotic heart disease	9	4	(44)	1	(12)	4	(44)
Generalized Arteriosclerosis	17	8	(47)	4	(23)	5	(30)
Family history of any of the above	21	6	(28)	11	(52)	4	(20)
Cirrhosis of liver	11	0	(0)	10	(91)	1	(9)
Normal volunteers	18	6	(33)	12	(66)	0	(0)

*Intermediate—O. D. above 1.0 at end of infusion, below 0.3 after 8 hrs.
**Slow—O. D. above 1.0 at end of infusion, above 0.3 after 8 hrs.
***Fast—O. D. below 1.0 at end of infusion, below 0.3 after 8 hrs.

TABLE I

tone bodies by a modification of the orginal method of Greenberg and Lester.*[3] These times were chosen not only to conform to previous clearing studies, but because preliminary work showed that they represented the peak and return toward normal in most patients. At the same time that blood ketones were done, the optical density of the serum was also measured by previously described methods, to assess the rapidity of clearing.

For the serum lipid study, 10 conscientious objectors on the metabolic ward at the United States Army Medical Nutrition Laboratory, Fitzsimons Army Hospital, Denver, Colorado were divided into two groups of six and four each. The group of six was subdivided into two groups of three, one group receiving 600 cc. of Lipomul intravenously and the other 600 cc. of Lipomul intragastrically. One week later the procedure was reversed. Blood was withdrawn from each subject before, at the end, 8, 16, 24, 36, and 48 hours after the beginning of each experiment. The second group of four subjects each received 600 cc. of Lipomul intravenously each day for five consecutive days. On the first, third and fifth days, blood was withdrawn from each subject before, at the end, 8, 16, and 24 hours after the beginning of the daily infusion. On the final day 36 and 48 hour bloods were also drawn.

The blood was chilled immediately upon withdrawal, the serum separated at ordinary centrifugal force and aliquot specimens taken for analyses. The remainder of the serum was then spun at top speed in a highspeed Servall supercentrifuge for 45 minutes (approximately 18,000 rpm). The subnatant serum was then removed and analyzed. The whole serum and subnatant were analyzed for total fatty acid by the method of Smith and Kik,[4] total and free cholesterol by a modification of the Sperry-Schoenheimer method,[5] lipid phosphorous by the method of Fiske and Subbarow,[6] and neutral fat by difference (TFA - [CE + PL] = NFA).

The possible effect of diet was appreciated so that the subjects in the second group receiving the multiple infusions were fed a constant, known diet in which the Lipomul was substituted isocalorically for fat during the infusion period. All infusions were started in an overnight-fasting state.

Results

The values for blood ketones rose in each subject immediately after the infusion and returned toward normal after 4½ more hours in all except one patient (Figure 3).

Effect of 600 cc of An Intravenous Infusion of A Fat Emulsion On the Blood Ketone Level in Eleven Subjects.

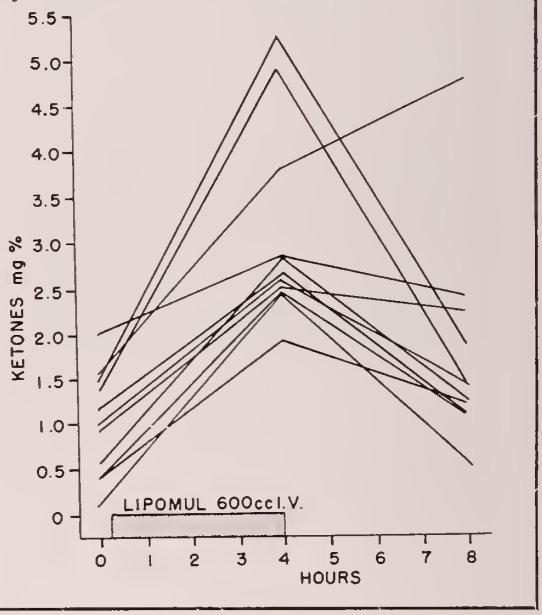


Figure 3.

However, it was surprising that the rise was so small considering the amount of fat, namely 90 Gms, infused so rapidly. These rises really are quite small, perhaps insignificant, but consistent. At attempt to correlate the optical density at the end of the infusion and the blood ketone at the same time results in an inconclusive graph (Figure 4). It should be noted that the patient with the highest ketone level was a very rapid clearer; however, the most rapid clearer was the poorest ketone producer. This dichotomy is explainable by the fact that it occurred in a severely ill patient with cirrhosis of the liver and impending hepatic coma in whom ketone production is notoriously inefficient. This type of experiment was abandoned because it was felt the small rises

*The author wishes to thank Doctor E. Werk, Chief Metabolic Division, Cincinnati Veterans Administration Hospital for performing the analyses for blood ketones.

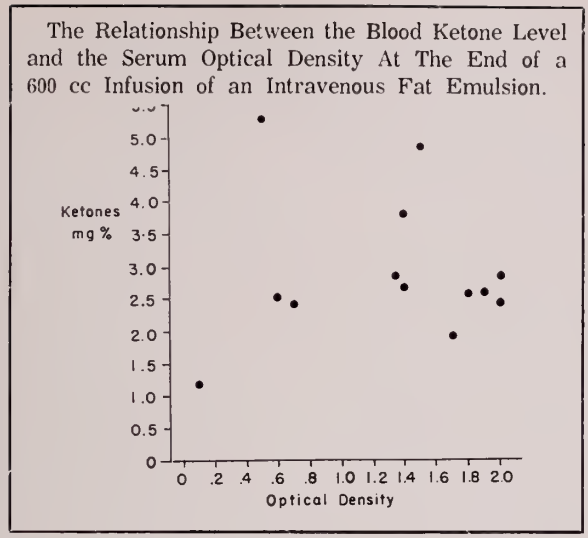


Figure 4.

in ketones following I.V. fat could not be used to access oxidation. Presumably under the conditions of the experiment, the oxidized 2-carbon fragments enter the citric acid cycle immediately so that acetone bodies do not have a chance to form. Therefore, other means must be sought to prove that rapid clearing of the blood necessarily means rapid oxidation of the infused fat.

The changes in serum lipids can best be described by referring to Figures 5 through 12. The subnatant values are interpreted as representing the lipid associated with solubilized fat or lipoprotein wheras the difference between whole serum and subnatant would represent the lipid mainly in the insoluble chylomicron and perhaps some very low density lipoprotein. There is a great quantitative difference between the effect on serum lipids of oral and I.V. Lipomul but very little qualitative differences. All changes are in the same direction. The single-infusion induced alterations in the six subjects in one group are similar to those in the four subjects after their first infusion.

The total fatty acids reach their peak in the whole serum immediately after the completion of the infusion and then rapidly return toward normal (Figures 5 and 6). The much lower peak in subnatant total fatty acids is reached four hours after that in whole serum and tends to be biphasic, rising again at 36 and 48 hours. The delayed rises in total fatty acids are interpreted as representing solubilized or lipoprotein increases consequent to the infused fat leaving the blood and then re-entering the circulation to be carried to the cell for either storage or energy. With repeated infusions there is a progressive rise indicating some tendency for slower clearing of the blood with successive infusions. Neutral fat patterns follow the total fatty acid picture quite closely (Figures 7 and 8).

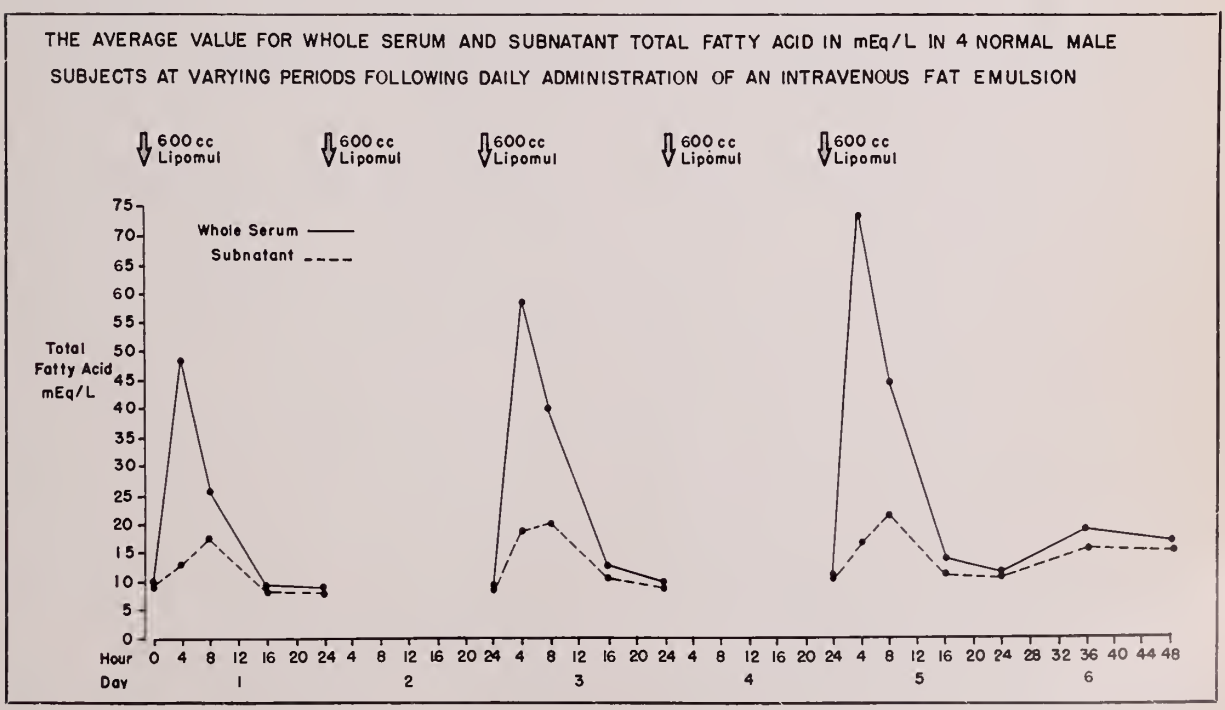


Figure 5.

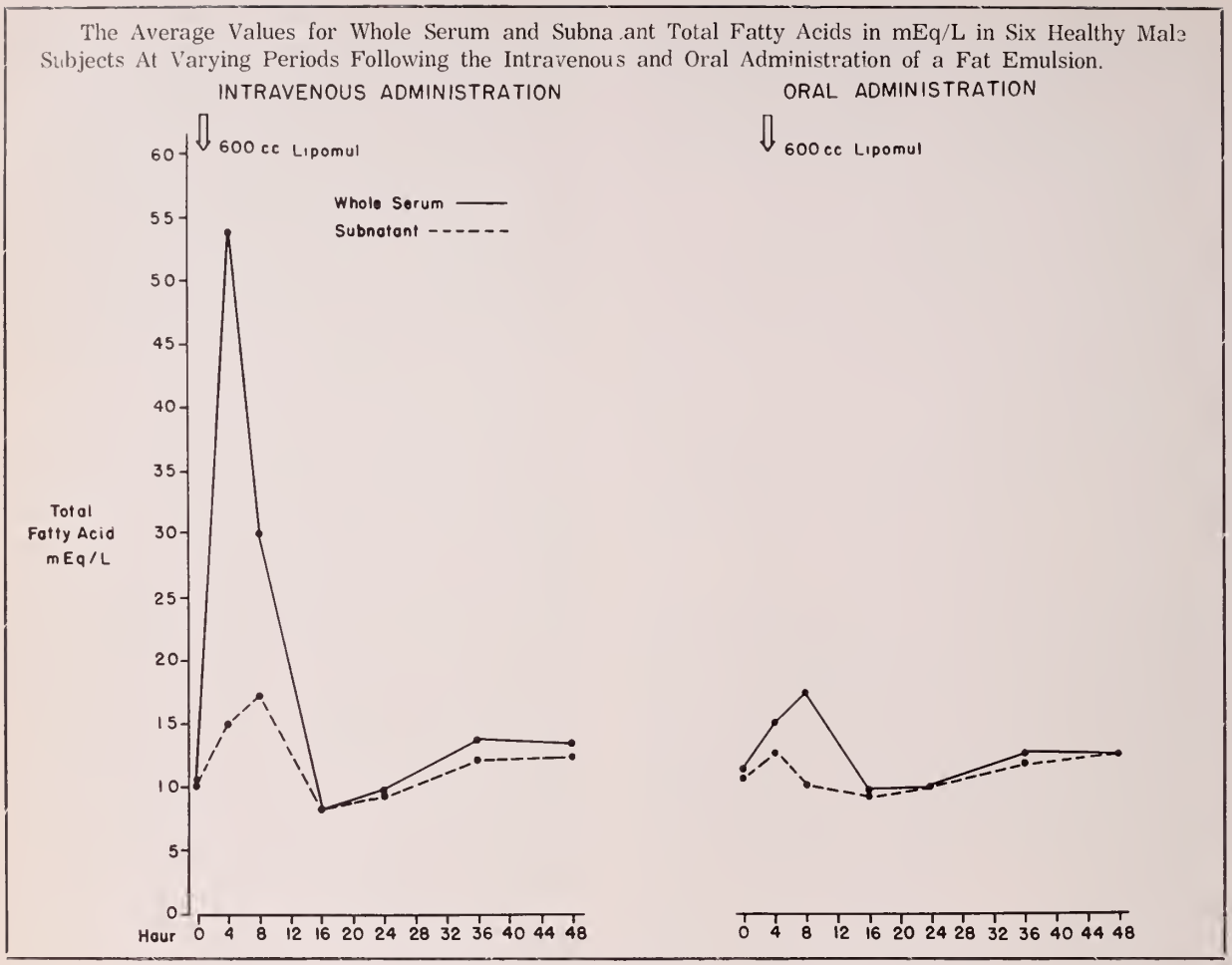


Figure 6.

The changes in the serum cholesterol were striking (Figures 9 and 10). With the initial infusion there is a slight rise in the whole serum total cholesterol reaching its peak either at 4 or 8 hours. The rise can not be accounted for by the Lieberman-Baurshard reacting material infused in the Lipomul. This must represent endogenous free cholesterol. Following the rise there is an abrupt fall, the lowest point occurring at 24 hours. The changes are entirely different in the subnatant, where the total cholesterol falls abruptly from the beginning. This difference between whole serum and subnatant suggests that serum cholesterol is moving into the chylomicron layer initially and then disappearing from the blood in this fashion. Similar but less dramatic changes occur following oral Lipomul. With successive infusions the above changes are intensified early, but by the fifth infusion very little total diminution occurs. Following discontinuation of the infusions there is an immediate rise toward pre-infusion levels. It will be seen from the graphs that all of the late fall in cholesterol is in the esterified fraction, while the early rise is in the free fraction.

The changes in lipid phosphorous were not especially unusual (Figures 11 and 12). The immediate rise was easily accounted for by the soybean phosphatide employed as the emulsifier in the emulsion. The fall seemed to follow the changes in total fatty acid.

Discussion

The above findings are preliminary and abbreviated. A more complete report is forthcoming. From previous observations concerning the clearing pattern of intravenous emulsions in various individuals, it is clear that what is found in these normal, healthy, young men may not necessarily be true in women or in older, ill patients. However, since so little is really understood concerning the pathways whereby fat is moved

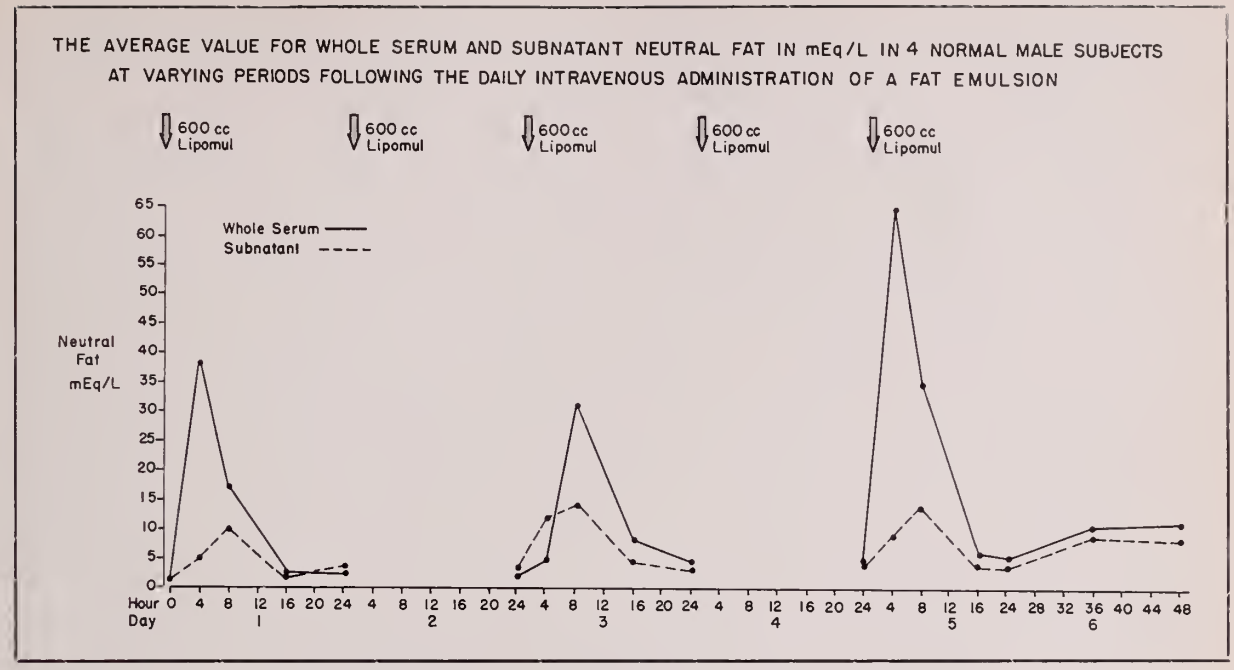


Figure 7.

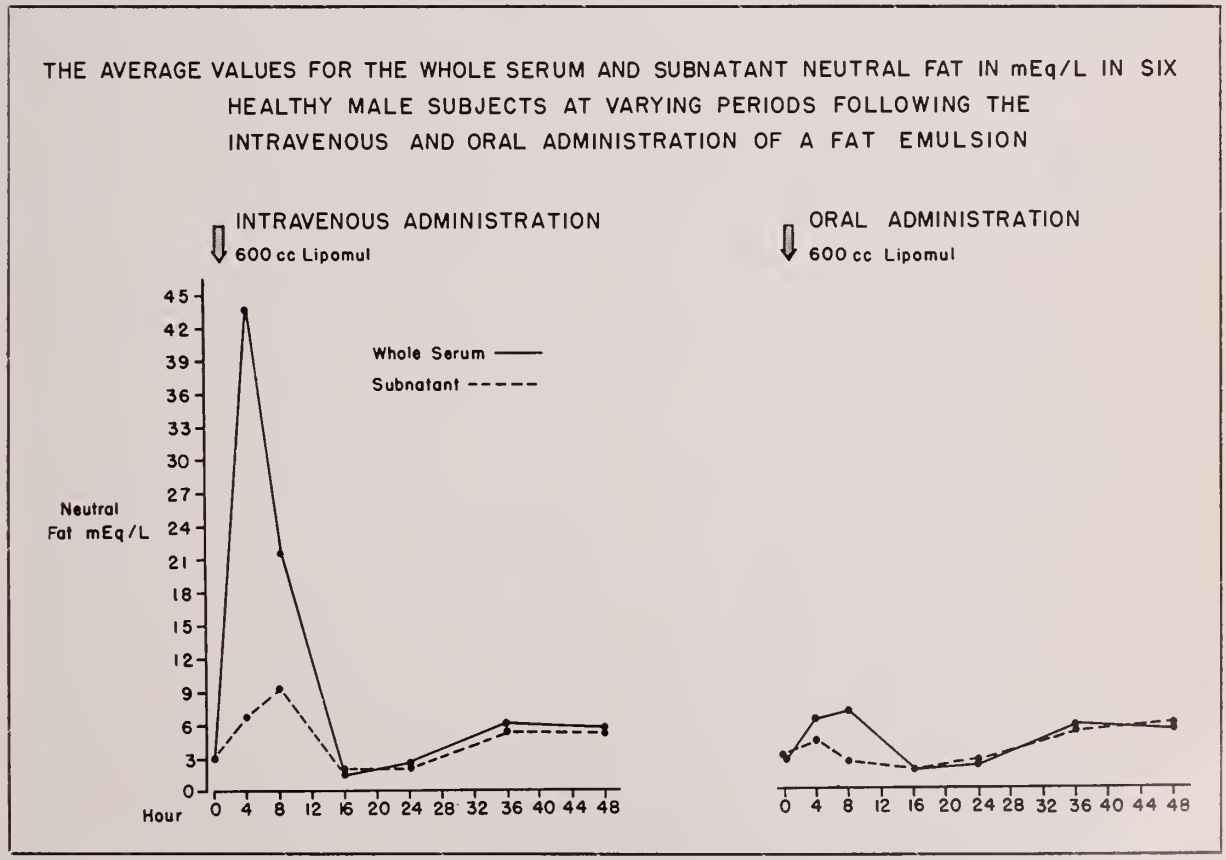


Figure 8.

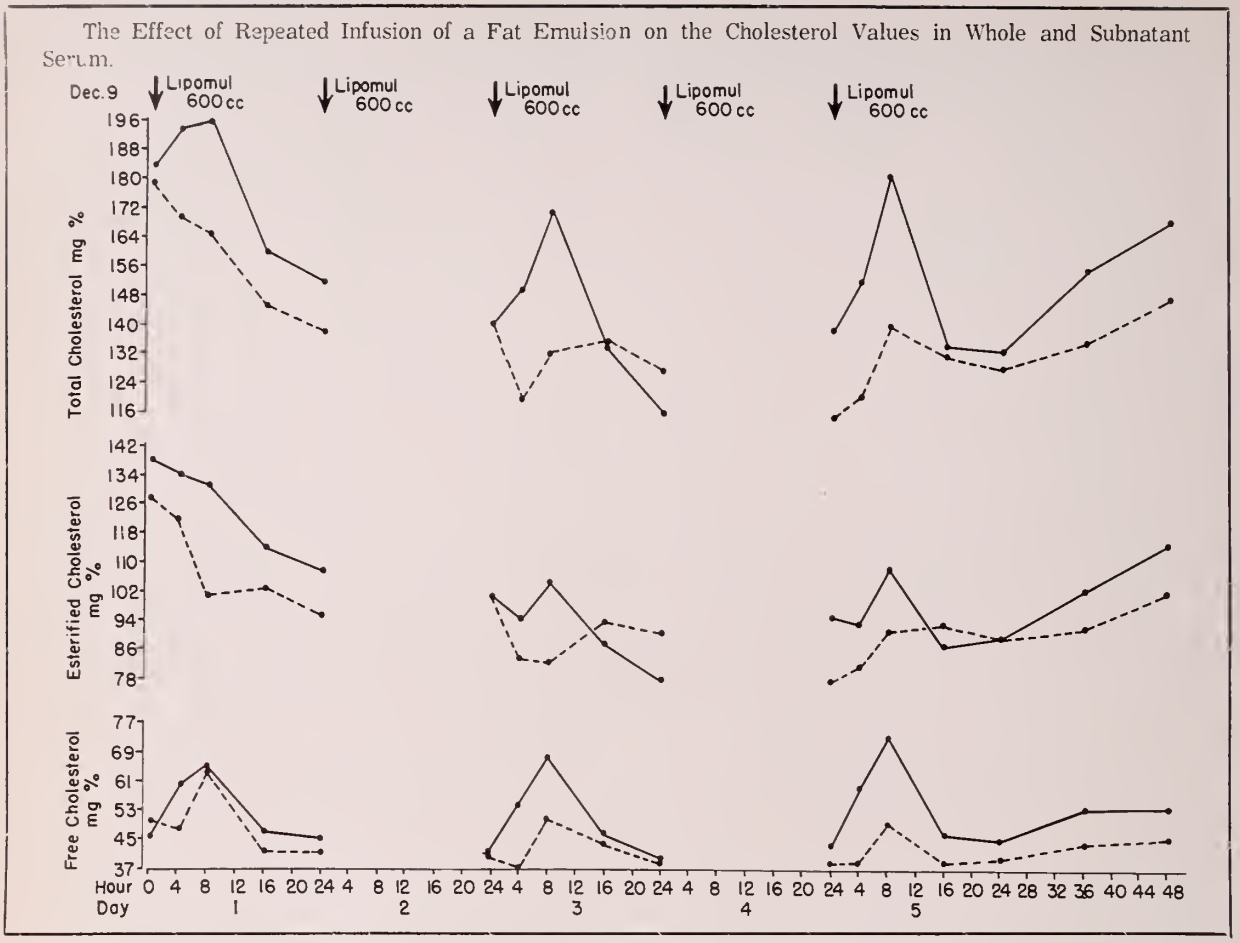


Figure 9.

from the blood to the liver and thence to the cell for energy or storage, certain hypotheses can be postulated from our observations. They are by no means proved.

The extent of intravascular vesus intracellular lipolysis has been argued for many years. The previously mentioned studies of Cleland and Iacono in which a very insignificant amount of "clearing factor enzyme" was found in the blood of our patients suggested that very little hydrolysis of the fatty acid chain occurred intravascularly. The very slight and delayed rise in subnatant total fatty acids following intravenous fat as compared to the rise in whole serum suggests that very little triglyceride fat is undergoing lipolysis in the blood but rather that it is being removed from the blood in the form of chylomicrons, hydrolyzed and then re-entering the blood in the form of lipoprotein to be transported to the cell. The most likely site for this to occur is in the liver. The secondary rise in total fatty acids that occurs in 36 or 48 hours probably represents a delayed increased production of lipoprotein consequent to the large fat load.

The daily increases in the height of the total fatty acids, following the infusion is explained by gradual overloading of the physiological clearing mechanism, whatever that may be. This phenomena of decreased tolerance to a fat load appears to be quite variable among individuals and may reflect the subject's need for energy. Thus, in our experience, patients who have a caloric need for the fat will clear their blood more rapidly and therefore have a greater tolerance in terms of accumulating fat in their blood at any given time than healthy individuals. Presumably prolonged hyperlipemia following fat infusions would be inadvisable, so that frequent observations of a patient's serum for lipemia is strongly urged in all subjects receiving multiple infusions.

The role of serum cholesterol in lipid

transport and metabolism has been a controversial subject. The immediate rise in whole serum free cholesterol suggests that the fat load called forth either stored or newly synthesized endogenous cholesterol. This cholesterol plus some of that already present in soluble lipoprotein moved readily into the unsoluble or chylomicron layer. The latter undoubtedly contains or is, in fact, very low density lipoprotein. This observation suggests that the high density lipoprotein can become low density lipoprotein perhaps within the blood stream merely by the addition of lipid molecules to its structure. Regardless of where this transportation is accomplished, cholesterol in the esterified form then leaves the blood stream with the chylomicron. The ultimate fate of a particular cholesterol molecule is not shown by these studies, but presumably the serum depression is very transitory since it starts rising 24 hours after the last infusion. It is interesting to speculate concerning the cholesterol-lowering potentials of such an emulsions since it contains a highly unsaturated oil, cottonseed oil. There is an increasing amount of data in the literature concerning the cholesterol-lowering potentiality of dietary unsaturated fat in humans.[7] It would be dangerous to relate acute changes such as we are reporting to prolonged effects. The acute effects can be reproduced by the emulsifier or glucose alone, and therefore are not specific for the fat. Furthermore, in our experience there has been no consistent variation in total

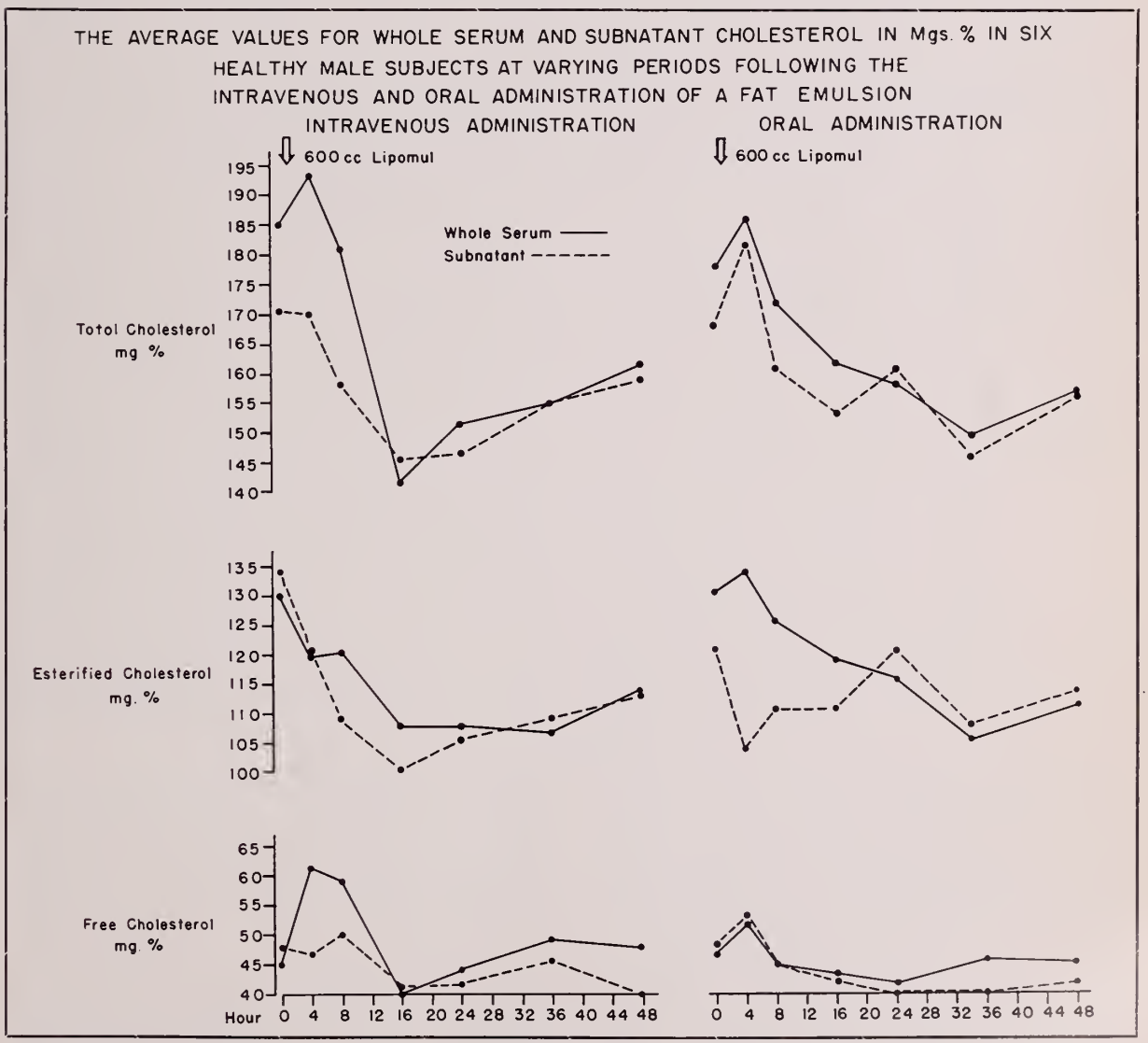


Figure 10.

The Average Value For Whole Serum and Subnatant Lipid Phosphorus in Mgs.% in Four Normal Male Subjects at Varying Periods Following the Daily Intravenous Administration of A Fat Emulsion.

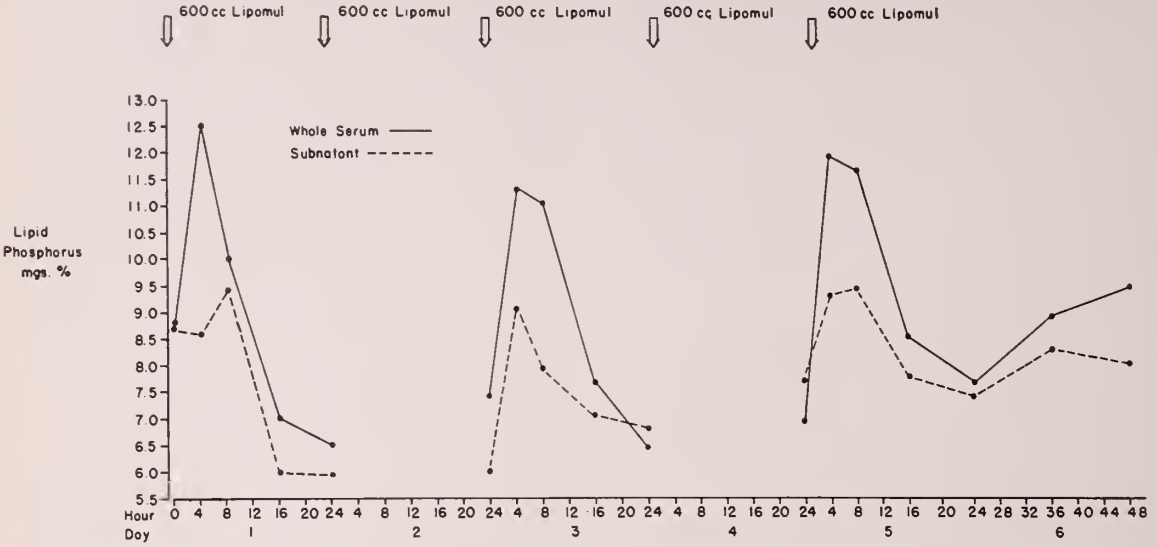


Figure 11.

The Average Values For Whole Serum and Subnatant Lipid Phosphorus in Mgs.% in Six Healthy Male Subjects At Varying Periods Following the Intravenous and Oral Administration of A Fat Emulsion.

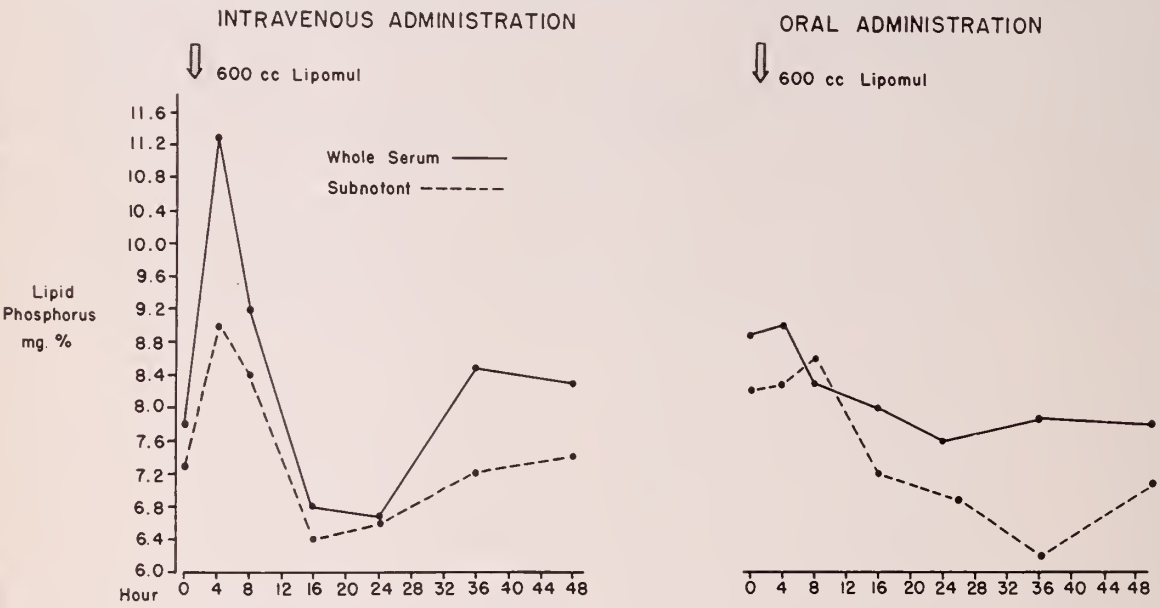


Figure 12.

cholesterol in patients receiving multiple infusions over a period of many days. That this should be expected is suggested by the observations in the present study that the changes in cholesterol on the fifth day of the experiment were strikingly different both in quantity and quality than those on the first day. There then appears to be some physiologic adjustment made to the repeated infusions. It would seem, therefore, to us that cholesterol depression by dietary unsaturated fat may involve other mechanisms than those observed in these acute experiments.

Mention has not been made of another important serum lipid; namely, the unesterified fatty acid fraction. Dole[8] and Gordon[9] have suggested that this fraction, which is

combined primarily with albumin, is the form in which the fatty acid is actually carried to the cell for oxidation. In the study performed prior to the present one, unesterified fatty acid in the serum was measured following a single infusion of Lipomul. As would be expected, it rose slightly at the end of the infusion and then promptly returned to normal. This is explicable on the basis of the tremendously rapid turnover of this fraction which has been demonstrated in animals.[10] Presumably this material disappears from the blood almost as rapidly as it enters. Therefore, an elevated level does not develop.

Many questions have been raised and very few answers given by these studies. Obviously much more careful work must be done not only in normals but in abnormals. The use of radioactive labelling technique will be helpful in tracing the pathways of lipid metabolism from the chylomicron to lipoprotein to the cell. It would appear, however, that the serum lipids play a major role in transport mechanisms.

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DISCUSSION

DOCTOR FURMAN: Doctor Mueller, the forced feeding of fat to experimental animals has been reported to produce a syndrome of widespread hemorrhage, including a pancreatitis, and this question asks if there is evidence that the pancreas might be involved in the syndrome which you have reported following lipomul?

DOCTOR MUELLER: We have not done any specific studies on pancreatic function. However, as I recall, Doctor Levenson did some serum amylase studies[1] in his patients which were normal. I can say that clinically there was no suggestion of acute pancreatitis. The pain, although present, was not of a severe nature. There were no abdominal findings which would suggest pancreatitis. I think it is a logical question to ask and probably should be looked at more in the future.

DOCTOR FURMAN: Pulmonary lesions have been reported following intravenous fat emulsion. Are these now being seen with the more recent preparations?

DOCTOR MUELLER: No, they are not.

DOCTOR FURMAN: Doctor Mueller, there is some evidence that clearing, or at least the transport of fat, is related to serum albumin and albumin levels; and furthermore, it has been shown that the degree of postprandial lipemia can be reduced by factors that increase carbohydrate metabolism, such as glucose. Would this suggest that the use of either or both of these agents, namely albumin and glucose, would speed clearing?

DOCTOR MUELLER: Of course there is glucose in the fat emulsion. The fat doesn't exert any osmotic pressure so that glucose must be added for this reason. There is 4 per cent dextrose in the fat emulsion. I don't have any information on the addition of more glucose to the fat emulsion. The question of albumin is an interesting one and one which we looked into a couple of years back. We checked to see what effect the addition of albumin to the fat emulsion might have on the incidence of adverse side effects. This was at the time that we were using the old emulsion and getting high incidence of reactions. Our studies at that time, which have been published,[2] showed that there was no significant change in the incidence of any of the side reactions after the addition of albumin. Unfortunately, at that time we were not studying the clearing mechanism, and I have not looked at that again since that time.

DOCTOR ARTZ: I would like to ask a question. How much intravenous fat emulsion would you give to a patient over a prolonged period and figure you were safe?

DOCTOR FURMAN: Doctor, Mueller, do you want to take that?

DOCTOR MUELLER: No, sir, this is a loaded question. I don't think anyone can answer that at the moment. I have adopted the arbitrary decision that I would stop after 10 injections and then wait awhile. I'm sure this is on the low side. We are at the present time studying one man who has received 32 or 33 infusions and still has not developed any difficulty. I only think, as I said, that people who are planning to use this material for long-term administration ought to be aware that these severe reactions can occur and should be prepared to stop the infusions with the first onset of any peculiarities in a patient. I just don't think we can give an arbitrary figure at this time as to how many infusions will produce this reaction.

DOCTOR FURMAN: Do you give this fat to people who have had myocardial infarcts within the past year?

DOCTOR MUELLER: If there is a clinical indication for it, yes. We have done some other studies using the ballistrocardiogram on persons receiving fat

emulsion. We did a group of normal subjects and then some patients with severe angina. We could detect no adverse effect on the ballistocardiogram during the fat infusion. I know of no incidents of coronary thrombosis that could be attributed unequivocably to the fat emulsion.

DOCTOR FURMAN: Doctor Abbott, do you have anything to add?

DOCTOR ABBOTT: Doctor Mueller's statements that he has an undernourished patient who received fat emulsion intravenoustly for 30 days is of great interest to me. We have given the same emulsion to patients for 12 to 15 days and to one individual for 18 days. We have not seen any of the so-called long-term serious reactions. In every case, the patients had a need for calories and nitrogen. It still makes one wonder if the patients who had serious reactions were not overloaded, especially when they were given 50 to 80 calories and three or more grams of fat per kilogram of body weight per day in a relatively inactive normal individual.

DOCTOR MUELLER: That is, as I said earlier, a very attractive theory. I think it is certainly possible that fat may appear in the tissue before it appears in the blood. The information we have on that is the one liver biopsy we did on this patient at the time she had a reaction. We could not demonstrate any increase of lipid in the liver.

DOCTOR ARTZ: This theory isn't quite so attractive to me. It so happens that we have had three patients on a minimal caloric intake who received large quantities of fat. All were very depleted patients and bled rather profusely. The coagulation times did not go quite as high as Doctor Mueller's patient, in the neighborhood of 24 to 26 minutes. Were your clotting times in silicon tubes?

DOCTOR MUELLER: Ours were siliconized.

DOCTOR ARTZ: This bleeding phenomena did occur in three patients who were depleted individuals on inadequate caloric intakes. However, our infusion of fat was a little more rapid than Doctor Mueller used. We used two infusions of fat a day. There is ample evidence that not all patients bleed because there are two burn patients at the Brooke Army Medical Center who have received 60 units of fat emulsion over a 30-day period with no particular difficulty and no bleeding tendency. It is not every patient that bleeds. Further investigation is necessary.

DOCTOR ABBOTT: I think that fat overloading may still be an important factor in spite of the fact that Doctor Artz says his patients weren't overloaded. I would like to point out that if his debilitated patients weighed 50 kilograms, they were getting from the intravenously administered fat over 3½ grams of fat per kilogram of body weight, which would probably represent more fat per day than an adult could transport and metabolize. We should think in terms of calories or grams of fat per square meter surface area or per kilogram of body weight per day when we are discussing the amount we are giving, **not** in total volume or grams. We have had patients who weighed 40 kilograms and thus on a 1800 caloric diet were getting over 40 calories per kilogram. In most of us who weigh 70 to 80 kilo's, such a caloric intake would represent an inadequate intake. I would also like to emphasize that oral nutritional intakes have to be considered in conjunction with intravenous alimentation. In discussing the fat intake of Doctor Artz's patients, if an oral intake of 50 to 100 or more grams of fat were taken daily, this would increase the total daily fat intake per kilogram of body weight.

DOCTOR FURMAN: Doctor Mueller, can a humoral pyrogenic factor be demonstrated in the blood of patients having a febrile reaction to intravenous fat? And the second question is what is the response of patients with nephrosis to intravenous fat?

DOCTOR MUELLER: That is relatively easy to answer, because I don't know the answer to either one. I don't know of any studies that have been done on the blood of patients receiving intravenous fat which would indicate a pyrogen. Whether or not a pyrogen exists is still open to some question. The fat emulsion itself is pyrogenic as tested in laboratories, according to the standard techniques for testing for pyrogens. Before I will accept the pyrogenic concept of the fever from fat emulsion, future work will have to be done.

I do not have any information on the use in nephrosis. We have used it in patients with uremia, but none with the nephrotic syndrome.

DOCTOR FURMAN: Doctor Mueller, would you care to speculate regarding what role, if any, the lipid mobilizing factors might be playing in these phenomena which you have reported this morning?

DOCTOR MUELLER: This would have to be strictly speculation because I have not had the opportunity to have any of this material to study. This material which has been found in high concentrations, particularly in the urine of horses given large amounts of cortisone, when injected into fasting animals, produces hyperlipemia. It originates from the anterior pituitary and its relationship, for instance, to growth hormones, which can also produce hyperlipemia, is still unclear. I just don't think there is enough information from any source to say whether it plays any role in the clearing of these fat emulsions or not. As I say, this work has been done principally in animals by one group of investigators, to my knowledge.

MEMBER OF THE PANEL: Do you know of any specific electrolyte changes that occur during or following fat emulsion administration?

DOCTOR MUELLER: Yes, this has been studied principally, to my knowledge, by Doctor Becker[3] in Chicago and by the group in Boston also. There is a slight decrease in potassium associated with the intravenous infusion of fat, with no essential change in sodium or in pH or CO_2.

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The Use of Fat Emulsion, Alcohol and Protein Hydrolysates in Surgical Patients

WILLIAM E. ABBOTT
HARVEY KRIEGER
STANLEY LEVY

THERE ARE NUMEROUS FACTORS which influence the metabolic response to operation as measured by the nitrogen, sodium and potassium balance. These include the age and the sex of the patient, the presence or absence of complications, the type and magnitude of the surgical procedure, and the nutrition of the patient. In order to study the effects of nutrition on the metabolic response to surgery it is necessary to remove as many extraneous complicating factors as possible. In the present work we have used only male patients who are undergoing hemigastrectomy in order to reduce the number of variables in the study.

Figure 1 summarizes the data obtained from three patients who were maintained by the intravenous administration of hexose solutions on the day of operation and for at least the next four postoperative days. E.W. received protein hydrolysates on the day of operation. In these and subsequent charts the vertical dotted line represents the day of operation. The areas below the zero line in the water, sodium, potassium and nitrogen balance charts represent the degree of negative balance, while the black areas above the zero line represent positive balance. In the use of the caloric intake the clear areas represent an intravenous intake while the cross hatched areas represent periods of oral diet. All three patients developed large negative nitrogen balances. During the five day period (day of operation and the next four days) the cumulative nitrogen deficits were 57, 70 and 63 gms. for patients E.W., D.B., and M.T. respectively.

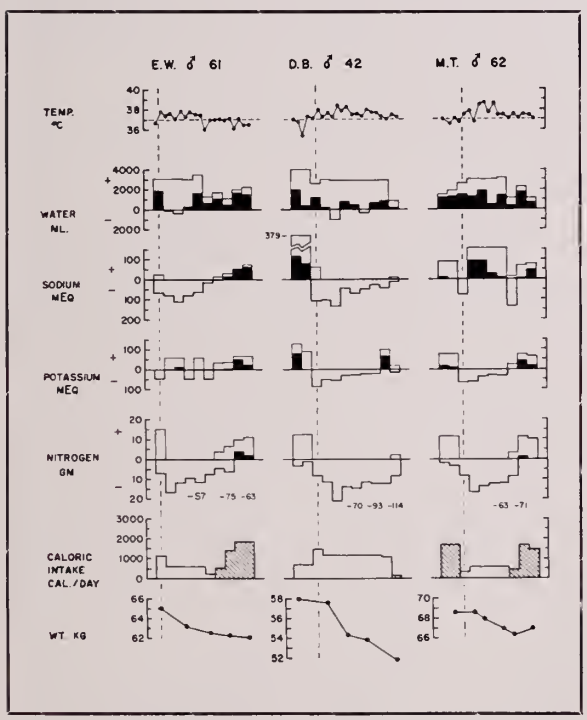


Figure 1. The balance data obtained from the study of three patients undergoing gastrectomy. On the day of operation and for at least the first four postoperative days they were maintained on nitrogen free intravenous regimen which provided 750 to 1000 calories.

The figures listed under the nitrogen balance charts show the cumulative nitrogen balances. Two of the patients (E.W. and M.T.) went into positive nitrogen balance when their caloric and nitrogen intakes increased. During the periods of intravenous feedings these three patients received no nitrogen and usually less than 1000 calories per day. One would not expect a patient who receives no nitrogen to be in anything but negative nitrogen balance. These patients were showing average daily deficits of 7 to 14 gms. of nitrogen during the first five day period on the intravenous regimen. In an earlier study[1] it was shown that normal non-operative male subjects maintained on an oral diet similar in caloric and nitrogen content developed a daily nitrogen deficit of approximately 7 gm. Calloway and Spector[2] have also reported that individuals on diets devoid of nitrogen and receiving 750 or more calories per day were in negative balance to the extent of 7 to 8 gms. per day. They also reported that with smaller caloric intakes greater nitrogen deficits occurred. These three postoperative patients showed nitrogen deficits similar to or a little greater than one would expect in non-operative patients maintained on comparable regimens. During the time the patients were in negative nitrogen balance D.B. and M.T. who received no potassium were also in negative potassium balance. The potassium bal-

ance of E.W. varied depending on his intake of potassium. When patients D.B. and M.T. were given potassium they immediately went into positive potassium balance. Patient D.B. who had the largest nitrogen deficit also had the greatest change in body weight (6 kg.) although the other two patients had lost weight to the extent of 2 to 3 kg.

Figure 1 illustrates that the patients who were maintained on intravenous regimens consisting only of hexose solutions with or without added electrolytes developed large nitrogen deficits. Such regimens are devoid of nitrogen and deficient in calories offering usually less than 1000 calories per day. With such an abnormal nutritional intake the patients lost between 3 to 5 kg. of body weight during the course of the study. With the development of various nutritional products (protein hydrolysates, fat emulsions, etc.) suitable for intravenous administration it is no longer necessary to maintain the postoperative patient on a starvation regimen.

Figure 2 summarizes the data obtained from three patients who were maintained during their postoperative periods on intravenous regimens consisting of protein

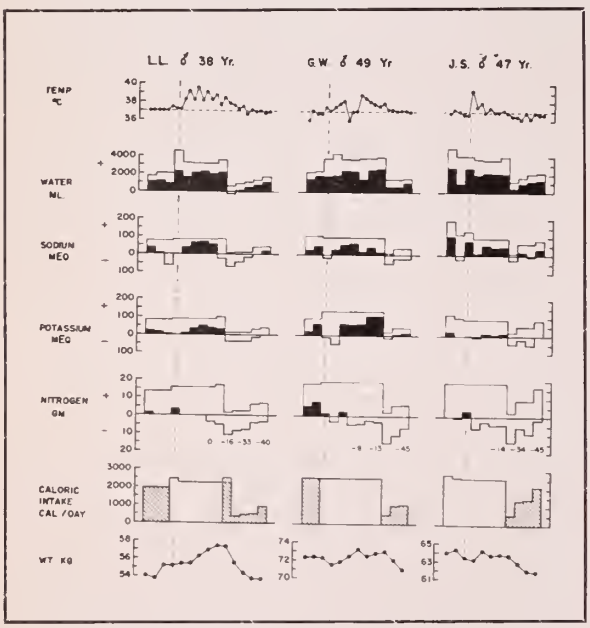


Figure 2. The balance data are shown from the study of three patients who had gastrectomies. The day of operation and at least the first four postoperative days these patients were maintained on intravenous regimens which provided 12 to 18 grams of nitrogen and 2500 calories.

hydrolysates containing sugar and electrolytes. They were also given a fat emulsion as a source of extra calories. With such a regimen it is possible to give between 15 and 18 grams of nitrogen (equivalent to 93 to 100 gm. of protein) and 2500 or more calories per day. During the five day period consisting of day of operation plus the first four postoperative days, patient L.L. showed a cumulative nitrogen deficit of 0.5 gm., G.W. 13 gm. and J.S. 14 gm. It should be noted that larger nitrogen deficits developed when the patients were switched from the high caloric, high nitrogen intravenous regimen to an oral diet which was lower in nitrogen and calories. Usually the reason for the development of these deficits was that the intake of nutrients was not adequate to maintain nitrogen equilibrium when the patients were first switched to an oral diet.

It should be also noted that these patients were retaining potassium during the immediate postoperative period. During this time they were receiving 100 to 120 mEq. of potassium per day. Patent L.L. had the minimal (0.5 gm) five day cumulative nitrogen deficit postoperatively in spite of the fact that he developed a fever which reached 40°C at times. One would normally expect that a fever would cause an increased breakdown of body tissue and an increased nitrogen deficit.

During the period of intravenous feedings these patients either maintained their body weight or gained small amounts. Part of this gain could be attributed to the retention of both sodium and water.

From the data presented in Figure 2 one might conclude that following major surgery (e.g., gastric resection) that patients need not show large nitrogen and potassium deficits. In earlier studies[1-3] we have shown that the nitrogen deficit in a postoperative patient is dependent upon the nitrogen and caloric intake. With increasing amounts of calories and nitrogen the patients show smaller nitrogen deficits. Giving 12 to 18 gm. of nitrogen by the parenteral route is a simple procedure with the modern protein hydrolysates but attaining an adequate ca-

loric intake by this route is difficult in all but patients of small stature if one does not employ fat emulsions. It has been shown[4] that the dog utilizes intravenosly administered fat emulsions as well as orally administered fat. It has also been noted[5] that traumatized (multiple fractures of one leg) rats do not handle intravenously administered fat in a manner different than that which occurs in the normal rat. By increasing the carbohydrate content of protein hydrolysates it is possible to increase the caloric content. But increasing the carbohydrate content beyond about 12½ per cent renders the solutions too hypertonic to be used without extreme precautions (very slow administration in large veins, etc.). The caloric content of an intravenous regimen may also be increased by adding alcohol solutions to protein hydrolysates. Patient R.R. was given a 5 per cent protein hydrolysate which contains 12.5 per cent fructose and 2.4 per cent alcohol. Each liter of this solution provides 800 calories.

There are reports in the literature[6,7,8] which indicates that the anabolic hormone testosterone and its derivatives tend to produce positive nitrogen balance in postoperative patients being maintained on adequate amounts of nitrogen but suboptimal caloric intakes. Figure 3 indicates data obtained from three different individuals who had a subtotal gastric resection. They were maintained on intravenous regimens for at least the day of operation and the first four postoperative days. In some cases the patients received intravenous alimentation preoperatively. They had nitrogen intakes of 12 to 18 gm. per day and caloric intakes ranging between 2200 and 2500 calories per day. In addition each patient received a day or two preoperatively 250 mg. of a long acting testosterone derivative, depo-nortestosterone or depo-testosterone. These individuals showed cumulative nitrogen balances of from -15 to +10 gm. for the five day period of study. Again it may be noted that when patients C.N. and R.R. were switched to an oral diet they developed larger cumulative nitrogen deficits than those which occurred during the immediate postoperative period. This was due to the fact that both their caloric and nitrogen intakes were too low to maintain nitrogen equilibrium during the initial oral feedings. It cannot be definitely decided from these studies whether or not the anabolic hormone produces a substantial reduction in the nitrogen deficit in patients receiving adequate amounts of calories and nitrogen. The increased caloric intake in these patients was produced by administering protein hydrolysates containing carbohydrate and a fat emulsion. During the immediate postoperative period the patients showed either very little change in body weight or actually a gain. The weight gain exhibited by these patients is due primarily to the retention of sodium and water.

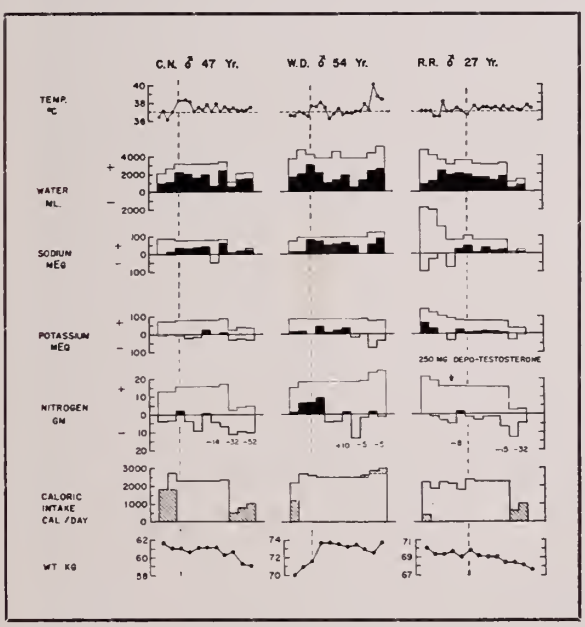


Figure 3. The balance data obtained from the study of three patients who had gastrectomies. They were given an injection of a long acting testosterone derivative 1 to 2 days preoperatively. C.W. and W.D. received 250 mg. of depo-nortestosterone cyclopentylpropionate and R.R. depo-testosterone.

The studies shown in the preceding three figures were carried out in postoperative patients who did not have complications. It is our belief that postoperative complications, e.g., hemorrhage, infection, atelectasis, etc., may cause greater nitrogen deficits than the operative trauma. Figure 4 shows the data obtained from the study of two patients who had postoperative complications. Patient F.R. had postoperative hemorrhage and pneumonitis. During the day of operation and the next four postoperative days, he developed a cumulative nitrogen

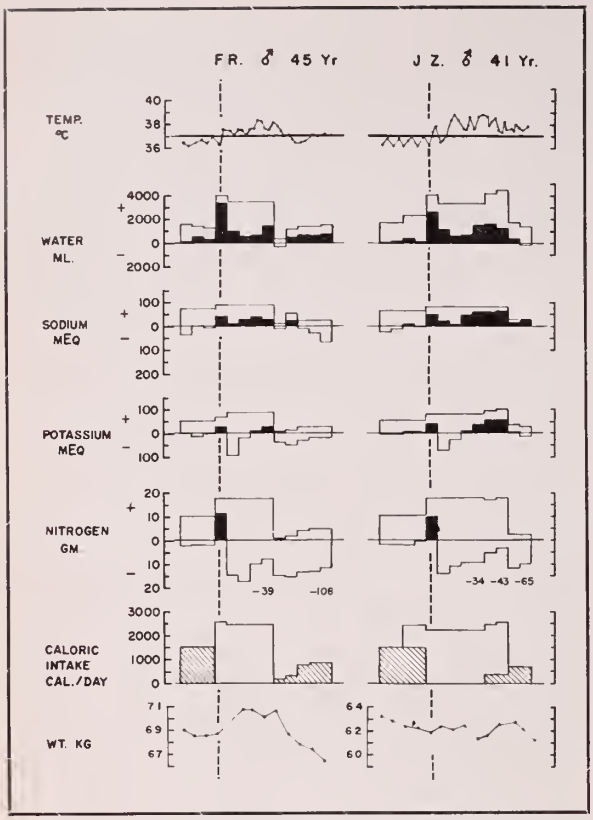


Figure 4. Shown are the balance data obtained from the study of two patients maintained on nutritionally good intravenous regimens postoperatively but developed complications.

deficit of 39 gm. in spite of receiving 2500 calories and about 18 gm. of nitrogen daily. J.Z. had a wound infection, atelectasis, and showed a 34 gm. postoperative five day cumulative nitrogen deficit in spite of an excellent nitrogen and caloric intake. Both of these patients showed large cumulative nitrogen deficits when they were switched to oral regimens. The nitrogen deficits of these patients should be compared with those in the previous two figures. From the present work and earlier studies it might be concluded that the major portion of the nitrogen deficit which occurs postoperatively in the uncomplicated case is due to the poor nutritional intake with which the patient is provided.

It has been shown definitely that the nitrogen and potassium deficits which occur in postoperative patients can be minimized or even negated by adequate nutrition during the pre and postoperative periods. However, one cannot show by the usual criteria that the patient is clinically better because he received a nutritionally adequate regimen postoperatively. By the same token it has never been demonstrated that starvation per se is advantageous. On the other hand it is well known that adequate nutrition during the pre and postoperative periods can reduce the morbidity and mortality rates of debilitated and malnourished surgical patients.

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DOCTOR FURMAN: Doctor Abbott, in any given patient, how do you decide what portion of the day's caloric intake shall be derived from fat, protein, or carbohydrate, or should it be mainly from one?

DOCTOR ABBOTT: Before answering your question, I would like to say that reports from good laboratories have indicated that nitrogen was not spared in patients who were given a fat emulsion intravenously, postoperatively. However, in these studies the patient did not get any nitrogen; obviously, it can't be spared if none is given. We have given 2,000 nitrogen-free calories to patients who weighed 50 or 60 kilograms and they waste nitrogen to about the same extent that they would if they had been given only 400 to 600 calories. It is obvious, first of all, as far as nitrogen balance and good nutrition is concerned, that you need protein. We feel that protein should make up about 20 to 25 per cent of the total caloric intake in adult patients following injury or in the postoperative state. I think everyone agrees a little higher intake of protein is probably desirable in patients who are repairing damaged tissues. As to the other source of calories, we feel that sugar is readily available for energy utilization and fat more slowly utilized, so that we prefer to give about half the non-protein calories as carbohydrate and the remainder as fat. This means fewer grams in the form of fat are taken since one gram of fat supplies nine calories to only four calories for a gram of sugar. We have not done any studies in patients who were maintained on a diet totally made up of just protein and fat. Actually, as Doctor Mueller has emphasized, we could not do this with the present fat emulsion because four or five per cent sugar is contained in the available emulsions.

DOCTOR FURMAN: Do you hesitate to infuse protein hydrolysates if there is any degree of uremia or renal insufficiency?

DOCTOR ABBOTT: I think if there is a renal shut-down, that a very minimal intake of nitrogen or a zero nitrogen intake is probably desirable. There have been studies by Doctor Ole Malm, now at Walter Reed Hospital and formerly from Oslo, Norway, in patients who had a renal shut-down which showed that two to three gram intakes of nitrogen per day did not exceed the wear and tear nitrogen and thus such an amount was well tolerated. Anything above that would probably not be desirable. We believe that two or three liters of the protein hydrolysates are indicated (which would provide 12.5 to 18 grams of nitrogen) in adult patients who have fairly good liver and kidney function. Again, I think it should be emphasized that in the patients with severe liver disease, a high intake of protein may produce hepatic coma and serious effects.

DOCTOR FURMAN: Does the over-all nitrogen balance appreciably influence wound healing? How much may one speed recovery by maintaining the nitrogen balance strongly on the positive side?

DOCTOR ABBOTT: I believe that when there is marked protein depletion there is interference with wound healing. However, the wound seems to get priority in the utilization of proteins. Studies have been done which indicate that proteins will even be taken from muscle and liver to heal a wound. Doctor Carl Moyer and his group did some interesting studies in burned rats. They gave comparable caloric intakes with very low nitrogen intakes to some animals and good protein intakes to others, and the rate of wound healing after a standard burn was essentially the same. However, after 60 to 70 days, when the wounds were healed, the animals were autopsied and marked fat infiltration of the liver, or cirrhosis, was evident in the animals who had the low protein intake while the other animals apparently had normal livers. I believe that it is hard in this day and age, when we have minimal losses and apparently well-nourished patients to operate upon, to note any great difference. However, many years ago, during Napoleon's time when the Russians and French were fighting, the Russian Army was given vodka and good quantities of meat in their diets and their injured soldiers had a much lower mortality and morbidity rate that the other armies reported. On further study, it was found that when the caloric and nitrogen intakes in this latter group were tabulated, the injured men had poor nutritional intakes. So I think there is a relationship, but it is certainly not apparent in the well-nourished patient who had minimal difficulties.

DOCTOR FURMAN: What is the metabolic effect of alcohol? Is it all utilized calorically? What is the caloric value of alcohol? Is it the same as sugar?

DOCTOR ABBOTT: I believe that alcohol is utilized primarily for energy, although it may be metabolized and converted into sugars and fats within the body. The caloric value is around six to seven calories per gram of alcohol, four per gram of sugar and protein, and around nine calories per gram of fat.

DOCTOR FURMAN: Is not the problem with alcohol the question of the rate of administration?

DOCTOR ABBOTT: It has to be given relatively slowly to prevent side effects, and this limits one as to how many calories they can give from alcohol in 24 hours. We use only a two and a half to three and a half per cent alcohol solution and govern the rate of administration more by the sugar content rather than the amount of alcohol, so that we get little or no spillage of sugar into the urine. We have not noted any ill effects from alcohol and in many instances most of the patients have not known that they had been given it. As I stated, we govern our rates of infusion by the carbohydrate content of the solution and size of the patient because this is the nutrient most apt to be spilled in the urine. We try to give it at a rate not faster than 0.5 to 0.7 gm. of sugar per kilogram of body weight per hour. It can be given a little faster (0.85 gm per kil. per hour) in non-traumatized, afebrile patients.

PRESIDENT'S LETTER



In this first effort at composing a President's Letter, I wish to express my appreciation and admiration for the calm, rational manner in which this office has been administered during the past year by Alfred T. Baker, M.D., of Durant. Many of the Council and Committee appointments will be carry-overs of men carefully selected by Doctor Baker. Much of the work to be done will be a continuance of efforts in the direction which he has indicated for us in the past year. His counsel will be asked in all important matters of policy decision for this organization.

The Oklahoma City meeting, May 2, 3, and 4, was certainly one of our very best State Meetings, and Doctor Charles Bielstein and Doctor Bert Keltz with their committee members can take real pride in the smooth functioning of all sections of this meeting.

The Association is in good shape. We own our own home debt free and have money in the bank. Operations have been kept within the budget successfully for the past several years. There are 1568 active, dues paying members, with an additional 125 Life and Honorary Members. We have 60 more doctor members than in 1959. Our Headquarters Staff is one of the best in the nation. The organization is run for doctors and by doctors. We're going ahead.

Walter E. Brown, M.D.

Walter E. Brown, M.D.
President

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ALDACTONE™

effectively extends the medical control of edema or ascites.

It introduces a new therapeutic principle in the treatment of...

CONGESTIVE HEART FAILURE • HEPATIC CIRRHOSIS
THE NEPHROTIC SYNDROME • IDIOPATHIC EDEMA

ALDACTONE introduces a new class of therapeutic agent, the aldosterone-blocking agent providing:

satisfactory relief of resistant or advanced edema even when all other agents, alone or in combination, are ineffective or are only partially effective.

A New Order of Therapeutic Activity

ALDACTONE acts by blocking the effect of aldosterone, the principal mineralocorticoid governing the reabsorption of sodium and water in the distal segment of the renal tubules.

By so doing Aldactone establishes a fundamentally new and effective approach to the control of edema or ascites, including edema resistant or unresponsive to conventional diuretic agents.

Further, because of its different site and mode of action in the renal tubules, Aldactone has a true, highly valuable synergistic activity when used with a mercurial or thiazide diuretic.

What Physicians May Expect of Aldactone

It is fully expected that Aldactone will change present medical concepts of the therapeutic limitations of managing edema. Many patients living in a greater or lesser state of edematous invalidism can now be edema-free. To others, gravely ill, Aldactone will be life-saving.

When used alone, Aldactone will produce a satisfactory diuresis in about half of those patients whose edema is resistant to conventional diuretic agents.

When Aldactone is used in a comprehensive therapeutic regimen, which includes a mercurial or a thiazide diuretic, a satisfactory diuresis and relief of edema may be expected in approximately 85 per cent of edematous patients *who would not otherwise respond.*

DOSAGE: For most adult patients the optimal dosage of Aldactone, brand of spironolactone, is 100 mg. four times daily. Aldactone should be administered for at least four or five days before appraising the initial response, since the onset of therapeutic effect is gradual when it is used alone. Aldactone manifests accelerated activity with greater response as early as the first and second days when used in combination with a mercurial or thiazide diuretic.

SUPPLIED: Aldactone is supplied as compression-coated yellow tablets of 100 mg.

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WALTER E. BROWN, M.D.
President



CLINTON GALLAHER, M.D.
President-Elect

Walter E. Brown, M.D., a native Tulsan, graduated from Washington University School of Medicine in St. Louis in 1935, following which he served rotating internships at the Kansas City General Hospital and the State University Hospital in Oklahoma City. He was Assistant Radiologist at St. John's Hospital in Tulsa in 1937-38, and then accepted an appointment as Graduate Assistant in Radiology at the Massachusetts General Hospital in Boston. He served as radiologist to the 167th General Hospital in the ETO during World War II and has practiced his specialty of radiology in Tulsa since his return from military service in November of 1945. Doctor Brown is a Diplomate of the American Board of Radiology and a Fellow of the American College of Radiology. He served as Delegate to the State Medical Association from Tulsa County for five years, has been Secretary-Treasurer of his Society, and was Vice-President of the Tulsa County Society last year. He has held varied committee assignments in the State Medical Association, including the chairmanship of the Medicare Committee.

Another seasoned member of the OSMA was elected to succeed Doctor Brown in 1961. Clinton Gallaher, M.D., Shawnee, graduated from Northwestern University Medical School in 1929, after receiving pre-med training and two years' medical education at the University of Oklahoma. He subsequently interned at Buffalo General Hospital and established a general practice in Shawnee before returning to Chicago for a residency in E.E.N.T.

In 1937, he established an E.E.N.T. practice in Shawnee and began a long career of service to his community and profession. He has been an OSMA Councilor for twelve years, a member of the Oklahoma Board of Medical Examiners for twelve years and Speaker of the House of Delegates for eight years. In his own county, he has served as President and Secretary of the local medical society.

Among other professional honors is his membership in the American College of Surgeons.

Seven Hundred Attend 54th Annual Meeting

The OSMA's 54th Annual Meeting provided a fitting climax to another successful year of association activities. More than seven hundred physician members registered for the meeting; attending the scientific lectures as well as the many related events which are traditionally offered. C. M. Bielstein, M.D. and Bert Keltz, M.D., served as General Chairman and Scientific Works Chairman, respectively.

Scientific activities were held in Oklahoma City's Municipal Auditorium, on May 2, 3 and 4. They were preceded on April 30 and May 1 by business meetings of the Council and House of Delegates.

Business Activities

New officers elected during the House of Delegates meeting are: Clinton Gallaher, M.D., Shawnee, President-Elect; J. Hoyle Carlock, M.D., Ardmore, Vice President; Mark R. Johnson, M.D., Oklahoma City, Secretary-Treasurer; Marshall O. Hart, M.D., Tulsa, Speaker of the House of Delegates; and, C. M. Hodgson, M.D., Kingfisher, Vice Speaker of the House of Delegates. Wilkie D. Hoover, M.D., Tulsa, and Joe L. Duer, M.D., Woodward, succeeded themselves as Delegate and Alternate to the American Medical Association.

In other actions, the House of Delegates:

- Approved the report of the Council on Professional Education which proposed an extensive postgraduate education program to be jointly sponsored by the OSMA and the University of Oklahoma School of Medicine.
- Heard without comment the report of the Council on Insurance, which significantly announced a forthcoming rate increase in professional liability insurance.
- Approved parts of the Council on Public Health Report, which delineated association responsibilities in providing health care for the aged at the state and local levels.
- Amended the Constitution and By-Laws to the extent that the removal of a physician's narcotic permit does not automatically result in the termination of OSMA membership. Disciplinary decision will rest with Council.
- Commended Grady F. Mathews, M.D., for his years of service as Commissioner of Health.
- Re-affirmed opposition to compulsory Social Security for physicians.
- Re-affirmed opposition to Forand-type legislation and announced opposition to legislative compromise in providing health care for the aged through taxation.
- Rejected a resolution calling for the resumption of a contractural agreement between the OSMA and the Medicare Program.

PRESIDENTIAL APPOINTEES, 1960-1961*

EXECUTIVE COMMITTEE

Walter E. Brown, M.D. ..... Tulsa
Alfred T. Baker, M.D. ..... Durant
Clinton Gallaher, M.D. ..... Shawnee
J. Hoyle Carlock, M.D. ..... Ardmore
R. Q. Goodwin, M.D. ..... Oklahoma City
Joe L. Duer, M.D. ..... Woodward

CHAIRMEN OF COUNCILS

Public Policy
J. R. Stacy, M.D., Oklahoma City

Insurance
Bruce R. Hinson, M.D., Enid

Socio-Economic Activities
Mark R. Johnson, M.D., Oklahoma City

Professional Education
R. R. Hannas, Jr., M.D., Sentinel

Public Health
Bert T. Brundage, M.D. Thomas

*Complete committee appointments will appear in a later issue of the Journal



Vice Speaker J. Hoyle Carlock, M.D., presides at the House of Delegates meeting

• Opposed H.J.R. 517, a proposed constitutional amendment which would permit Oklahoma to become indebted up to $35.5 million to provide capital funds for higher education.

• Referred for further study to the Council on Professional Education a resolution which recommended the creation of a scholarship fund for medical students.

• Passed a resolution which provided that the OSMA take an active, non-partisan part in local, state and national government, particularly in regard to the selection of qualified candidates for office.

Inaugural Ball

On Tuesday evening, Walter E. Brown, M.D., Tulsa radiologist, succeeded Alfred T. Baker, M.D., Durant, as President of the Oklahoma State Medical Association. The inaugural ceremonies were followed by dancing to the music of Paul Neighbors and his orchestra. Some 350 physicians and their attended the evening's activities.

In his remarks to the group, Doctor Baker paid tribute to the cooperation he had received during his term of office and expressed gratitude for the honor that had been bestowed upon him.

Doctor Brown presented himself with a personal challenge to live up to the standards set by his predecessors and, despite, today's grave problems, voiced optimism for the ability of organized medicine to continue its progress.

The Scientific Program

Fifteen outstanding guest speakers drew maximum audiences at general sessions held in the Zebra Room of the Municipal Auditorium. Highlighting the program were panel discussions on vascular disease, perinatal mortality and rehabilitation.

The latter portion of the program was attended by nearly two hundred physicians, physical and occupational therapists, as well as by Senators and Representatives of the State Legislative Committee on Rehabilitation. In addition, a Roundtable Luncheon on Monday was well attended, as was the Tuesday Luncheon featuring a presentation entitled "How Public Opinion Can Socialize a Profession or Industry." The OSMA hosted O.U.'s senior medical class at the Tuesday event.

One hundred technical and scientific exhibits completed the well-balanced program. In the field of scientific exhibits, committee chairman Edward R. Munnell, M.D., presented awards to the following: Scientific

Exhibit: 1st place, Joseph W. Kelso, M.D. and Joseph W. Funnell, M.D.; 2nd place, Ethan Allen Walker, Jr., M.D.; 3rd place, Averill Stowell, M.D. "Klinical Kaleidoscope" (bizarre cases): 1st place, R. Kent Alexander, M.D.; 2nd place, Samuel M. Glasser, M.D. and Rene B. Menguy, M.D.,; 3rd place, J. Neill Lysaught, M.D.

Complementing the OSMA program were nine specialty society meetings, the majority of which were held on Monday evening during the state meeting. Guest speakers for the OSMA program also appeared before the specialty groups.

Record-Breaking Golf Tournament

Three records fell during the annual golf tournament which was held at Twin Hills Country Club under the direction of Peter E. Russo, M.D. In the first place, the seventy-six registrants made it the largest contest in OSMA history; secondly, the low gross trophy was won with an even par score; and, lastly, a hole in one was scored by A. C. Lisle, M.D., Oklahoma City.

Trophy winners were: Edmond H. Kalmon, M.D., Oklahoma City—Low Gross (71); Lester Moline, M.D., Lawton—Handicap (65); Averill Stowell, M.D., Tulsa, Birdie Trophy (3); A. C. Lisle, M.D., Oklahoma City—Low Hole (hole in one); C. E. Woodard, M.D., Drumright—High Score (137); and, John W. Records, M.D., Oklahoma City—High Hole Score (12). In addition to the trophy winners, eight physicians made scores of 80 or less.

Woman's Auxiliary Meeting

Oklahoma medicine's right arm, the Woman's Auxiliary, held a concurrent meeting at the Skirvin Hotel to review last year's achievements and lay plans for the succeeding year. Mrs. Clifford M. Bassett, Cushing, turned over the auxiliary reins to Mrs. Virgil Ray Forester, Oklahoma City, and Mrs. W. P. Fite, Sr., Muskogee, was named as President-Elect.

Other officers elected were: Mrs. Milton L. Berg, Tulsa, 1st Vice President; Mrs. T. A. Ragan, Norman, 2nd Vice President; Mrs. C. W. Freeman, Oklahoma City, Secretary; Mrs. John W. Records, Oklahoma City, Treasurer; and Mrs. Louis S. Frank, Oklahoma City, Treasurer-Elect.

The 109th Annual A.M.A. Meeting: A Forum for 2,000 Top Medical Men

The 109th annual meeting of the American Medical Association will be a forum presented by some of the nation's top scientific brains.

Approximately 2,000 physicians, all outstanding in their field, will participate in presenting the scientific program of the meeting to be held in Miami Beach, June 13-17.

There will be two general scientific meetings in the Grand Ballroom of the Fontainebleau Hotel, and other lectures, symposiums, and panel discussions in the Fontainebleau, Eden Roc Hotel, and in the new, air-conditioned Miami Beach Exhibition Hall. Sessions on dermatology, being held jointly with the Society for Investigative Dermatology, will be in the di Lido Hotel.

The opening general scientific meeting, Monday afternoon, June 13, will begin with the Joseph Goldberger Lecture on Clinical Nutrition. Dr. Carl A. Lincke, chairman of the A.M.A. Council on Scientific Assembly, will preside at this meeting.

The lecture will be followed by a symposium on nutrition, including an address by Grace A. Goldsmith, professor of medicine, Tulane University School of Medicine, New Orleans, on "Highlights on the Cholesterol—Fats, Diets and the Atherosclerosis Problem."

The second general meeting will be a symposium on "Evaluation and Preparation of Patients for Anesthesia and Surgery," Tuesday morning, June 14, to which the sections on Anesthesiology, Diseases of the Chest, General Practice, Internal Medicine, Pediatrics, Pathology and Physiology, and Surgery have contributed. Participating will be Meyer Saklad, Providence, R.I.; Thomas Rardin, Columbus, Ohio; Eugene Turrell, Milwaukee, Wis.; John S. LaDue, New York City; Arlie R. Mansberger, Jr., Baltimore; George Meneeley, Nashville, Tenn.; Robert M. Smith, Boston and C. Rollins Hanlon, St. Louis, Mo.

Section Programs

What's new in surgery will be explored during a symposium and panel discussion on Wednesday morning. The program was developed from suggestions obtained from a survey of the heads of departments of surgery in the nation's medical schools.

The symposium will be on "Recent Advances in Treatment of the Cancer Patient" with an introduction to the problem including methods of decreasing spread of cancer cells during operations being presented by Warren H. Cole, Chicago. Doctor Cole also will serve as moderator of the panel on "Current Status of the Treatment of Advanced Cancer of the Thyroid and Breast."

Arthur M. Master, New York City, will speak on "Effort, Occupation (including physicians) in Coronary Occlusion" during a symposium on Medical Chest Emergencies, and John F. Briggs, St. Paul, Minnesota, will discuss "Pulmonary Embolism."

John H. Moyer, Philadelphia, will moderate a symposium on "Edema—Its Physiology and Use of Newer Diuretics in Its Treatment." Irving S. Wright, New York, will moderate a symposium on "Pathogenesis and Treatment of Thrombo-embolic Phenomena." Daniel C. Moore, Seattle, Washington, will take part in a session on "Newer Trends in Diagnosis and Treatment" discussing "Oxygen—The Rational Therapy for Systemic Toxic Reactions from Local Anesthetic Drugs."

There will be panel discussions on "Tumors of the Trachea and Bronchial Tree," with Paul H. Holinger, Chicago, moderator, and one on "Disseminated Diseases of the Chest," with Harold O. Peterson, Minneapolis, moderator.

Highlights of other section meetings include:

Nervous and Mental Diseases—E. S. Gurdjian, Detroit, "Critique of Occlusive Disease of the Carotid Artery and the Stroke Syndrome." Leo H. Bartemeier, Baltimore, "Comments on the Relations Between Psychiatrists and Other Physicians."

Obstetrics and Gynecology—Stirling G. Pillsbury, Long Beach, California, "31,595 Deliveries with One Maternal Death." Jerome M. Kummer, Santa Monica, California, "An Answer for Criminal Abortion."

Internal Medicine—"Symposium on Hypocholesteremic Drugs." Walter L. Palmer, Chicago, "The Billings Lecture: Causality in Peptic Ulcer." Rudolph H. Kampmeier, Nashville, Tennessee, "Collagen Diseases—Some Unanswered Questions."

Laryngology, Otology, and Rhinology—George A. Sisson, Syracuse, New York, "Problems and Complications in Head and Neck Surgery." C. M. Kos, Iowa City, Iowa, "Five Year Results of Stapes Mobilization and Current Results with Vein Plug Stapedeoplasty."

Preventive Medicine—Jan H. Tillisch, Rochester, Minnesota, "Medical Aspects of Safety in the Air." Robert B. Stonehill, San Antonio, Texas, "Air Travel and the Cardiopulmonary Patient," a film presentation.

Orthopedic Surgery—Sidney Keats, Newark, New Jersey, "Surgery of the Extremities in the Treatment of Cerebral Palsy." H. R. McCarroll, St. Louis, Missouri, "The Management of Complicated Ununited Fractures of the Tibia and Fibula."

Pediatrics—Leonard S. Sommer, Miami, moderator, "Symposium on Congenital Heart Disease—Present-day Status from Viewpoint of Practitioner of Medicine."

The section on Miscellaneous Topics will feature a half-day session on the "Management of the Older Patient." Participants include Philip Thorek, Chicago.

Exhibits Number 290

About 290 exhibits will be on display in the Miami Beach Exhibition Hall, representing the most outstanding exhibits selected from 540 applications.

A new special exhibit on Body Fluid Disturbances, offering a complete course in body fluids, will be presented in its entirety on two days. Different aspects will begin at specific times to permit physicians to attend only those parts in which they are particularly interested.



Pictured with Miss Mamie Richardson, first place winner of the Governor's Committee on Employment of the Handicapped essay contest are (left to right) Alfred T. Baker M.D., President of the Oklahoma State Medical Association, Durant; Len Yarborough, Executive Vice-President of the AFL-CIO, Tulsa; Roy Tillman, President of the AFL-CIO, Oklahoma City; Governor Edmondson; Miss Richardson; Mrs. Irene Mitchell, teacher of the first-place winner, Ringling; Lt. Governor Nigh; and James Reinmiller, President of the Oklahoma Association of Insurance Agents, Hobart.

Ringling Student Wins Governor's Essay Contest

Miss Mamie Richardson, senior at Ringling High School, placed first in the state essay contest, sponsored by the Governor's Committee on Employment of the Handicapped. More than 4,500 students wrote essays on "Jobs for the Handicapped—Passports to Dignity." Governor J. Howard Edmondson, Lt. Governor George Nigh, and members of the Governor's Committee met with the 10 state essay winners in the Blue Room of the state capitol, where Mamie and other winners were presented their awards.

Mamie received an expense-paid trip to Washington, D.C., to attend the meeting of the President's Committee, May 5-6. This annual award was presented by Len Yarborough of the Oklahoma AFL-CIO. Mr. James Reinmiller, President of the Oklahoma Association of Insurance Agents, presented a $200 savings bond. Mamie will also receive a two-year tuition and fee scholarship from Southeastern State College in Durant.

Mrs. Irene E. Mitchell, teacher of the first-place winner, received an expense-paid trip to accompany Mamie to Washington from Doctor Alfred T. Baker, President, Oklahoma State Medical Association, and a plaque from Lt. Governor George Nigh, which was sponsored by the employees of the Oklahoma City Goodwill Industries.



Shown congratulating Mrs. Irene Mitchell, teacher of the award winner, are Alfred T. Baker M.D., President of the Oklahoma State Medical Association, Durant (left), and Dick Graham, Executive Secretary, Oklahoma State Medical Association.

Carcinoma of the Skin Subject for May Symposium

Carcinoma of the Skin will be the subject of the Sixth Annual Surgery, Radiology, Pathology Symposium to be held May 27-28, 1960 in the auditorium of the University of Oklahoma Medical Center. Sponsored by the Office of Postgraduate Education, University of Oklahoma Medical Center, the Oklahoma Association of Pathologists, the Oklahoma State Radiological Society and the Oklahoma Chapter of the American College of Surgeons, the symposium will afford physicians two days of lectures, round table discussions and case presentations.

A group of specialists has been selected to attract the attention of surgeons, dermatologists, radiologists, pathologists and others interested in carcinoma of the skin problems. Guest speakers will include: Stephen R. Lewis, M.D., plastic surgeon, Galveston, Texas; Charles L. Martin, M.D., radiologist, Dallas, Texas; George T. Pack, M.D., neoplastic surgeon, New York City; Jose M. Sala, M.D., radiologist, Columbia, Missouri; Danely P. Slaughter, M.D., surgeon, Chicago, Illinois and J. Leslie Smith, M.D., pathologist, Houston, Texas.

A complete program follows:

FRIDAY, MAY 27 AND SATURDAY, MAY 28, 1960

FRIDAY MORNING, MAY 27

8:00 a.m. Registration

8:40 a.m. INTRODUCTION
John A. Schilling, M.D.

8:45 a.m. BENIGN AND PSEUDO-NEOPLASMS OF THE SKIN
Lymphoid tumors—hemangiomas of the skin
Lesions previously classified as malignant
Problems in histologic diagnosis and prognostic evaluation
J. Leslie Smith, Jr., M.D.

9:40 a.m. PRECANCEROUS LESIONS OF THE SKIN
Stephen R. Lewis, M.D.

10:10 a.m. Intermission

10:30 a.m. PRINCIPLES OF DIAGNOSIS OF SKIN TUMORS
Clinical evaluation on physical examination
Diagnostic maneuvers
Biopsy techniques and principles
Danley P. Slaughter, M.D.

11:00 a.m. ADNEXAL TUMORS OF THE SKIN
J. Leslie Smith, Jr., M.D.

11:30 a.m. ROLE OF RADIOTHERAPY IN THE TREATMENT OF BENIGN AND PRECANCEROUS LESIONS OF THE SKIN
Charles L. Martin, M.D.

12:00 noon Lunch

FRIDAY AFTERNOON, MAY 27

1:30 p.m. MALIGNANT SKIN NEOPLASMS — HISTOLOGIC DIFFERENTIATION AND BIOLOGIC BEHAVIOR
J. Leslie Smith, Jr., M.D.

2:00 p.m. PROBLEMS DEALING WITH THE PIGMENTED LESIONS INCLUDING MALIGNANT MELANOMA
George T. Pack, M.D.

2:30 p.m. BASAL CELL CARCINOMA AND PERSISTANT BASAL CELL CARCINOMA
Stephen R. Lewis, M.D.

3:00 p.m. Intermission

3:20 p.m. RADIOLOGICAL TREATMENT OF CANCER OF THE SKIN AND FACE
Charles L. Martin, M.D.

3:50 p.m. INDIVIDUAL VIEWS ON ABOVE SUBJECTS WITH DISCUSSION OR QUESTIONS AND ANSWERS FROM THE PANELISTS AND REGISTRANTS
Moderator: Stephen R. Lewis, M.D.
Participants: (All other guest lecturers)

5:20 p.m. End of Afternoon Session

7:00 p.m. SPONSORS SOCIAL HOUR AND BUFFET
Oklahoma Chapter of the American College of Surgeons
Oklahoma Association of Pathologists
Oklahoma Association of Radiologists

SATURDAY MORNING, MAY 28

8:45 a.m. MALIGNANT MELANOMA—HEAD AND NECK
George T. Pack, M.D.

9:30 a.m. INDICATIONS AND CONTRA-INDICATIONS OF ROENTGEN THERAPY IN CARCINOMA OF THE SKIN
Jose M. Sala, M.D.

10:00 a.m. INDICATIONS AND CONTRA-INDICATIONS FOR THE SURGICAL MANAGEMENT OF MALIGNANT SKIN TUMORS
Danley P. Slaughter, M.D.

10:30 a.m. Intermission

10:50 a.m. RADIOLOGIC TREATMENT OF SKIN CANCER OF THE NOSE, EAR AND ABOUT THE EYE
Charles L. Martin, M.D.

11:30 a.m. TECHNIQUES OF SURGICAL MANAGEMENT OF MALIGNANT SKIN TUMORS
Danley P. Slaughter, M.D.

12:10 p.m. Lunch

SATURDAY AFTERNOON, MAY 28

1:15 p.m. PATHOLOGIC CHANGES FOLLOWING TREATMENT OF MALIGNANCIES OF THE SKIN BY PERFUSION WITH P.A.M. DERIVATIVES
J. Leslie Smith, Jr., M.D.

1:45 p.m. MALIGNANT MELANOMA—TRUNK AND EXTREMITIES
George T. Pack, M.D.
2:30 p.m. GENERAL PLASTIC RECONSTRUCTIVE MEASURES
Stephen R. Lewis, M.D.
3:10 p.m. Intermission
3:30 p.m. INCIDENCE AND RADIOLOGIC TREATMENT OF LYMPH NODE METASTASIS
Jose M. Sala, M.D.
4:00 p.m. PANEL: TREATMENT OF CARCINOMA OF THE SKIN
Wrap-up questions and answers
Individual views of participants
Uncovered topics
Moderator: Danley P. Slaughter, M.D.
Participants: Stephen R. Lewis, M.D., Charles L. Martin, M.D., George T. Pack, M.D., Jose M. Sala, M.D.
2:00 p.m. J. Leslie Smith, Jr., M.D., with Oklahoma Association of Pathologists and registrant pathologists

Oklahoma's Reserve Hospital Is One Year Old

Oklahoma's first semi-mobile army reserve hospital, the 44th Evacuation Hospital, recently celebrated its first birthday. It was created by the Defense Department in April, 1959, as part of a reorganization of reserve forces.

Forty Men Needed

Colonel Hervey A. Foerster, M.D., Oklahoma City physician and hospital commander, pointed out his staff, which started with nineteen officers and eighty enlisted men, now includes thirty-eight officers, four nurses, three warrant officers and 116 enlisted men.

"However, the unit still needs about forty more enlisted men to be up to peacetime strength, and we can use some more doctors," the physician said. Physicians, he explained, may join the reserve unit even though they don't reside in Oklahoma City.

"We have one doctor who drives all the way from Ponca City for our drill sessions," Colonel Foerster said.

Summer Training Due

The 44th staff will take summer active training at Camp Hood, Texas, June 5-19. Half of the two week encampment will be spent in training at the post hospital. The second week will be spent in actual maneuvers, with simulated problems of battle conditions.

Wallis S. Ivy, M.D. Receives Double Honor



Shown receiving honors at the Stephens County Annual Doctors' Day Celebration is Wallis S. Ivy, M.D., (center), Duncan physician. Also pictured are (left to right) Mrs. Luther Roberts, Robert Dean Taylor, M.D., Luther Roberts, Doctor Ivy, Dwight Weedn, M.D. and Mrs. Ivy.

Wallis S. Ivy, M.D., Duncan physician, was twice honored when the Stephens County Medical Society held their Annual Doctors' Day celebration, March 29, 1960. A Fifty-Year Pin, commemorating his years of service to the medical profession was presented by the Oklahoma State Medical Association and, at the same time, a citation for outstanding accomplishment was pressented by the Duncan Chamber of Commerce.

Presentation of the Fifty-Year Pin was made by Dwight Weedn, M.D., President of the Stephens County Medical Society, and Luther P. Roberts of the Public Service Company presented the citation from the Chamber of Commerce.

Doctor Ivy is the oldest practicing physician from the standpoint of continuous service in Duncan and Stephens County. Coming to Oklahoma in 1915, Doctor Ivy established his practice in Marlow, moving to Duncan four years later. For the past 17 years, he has limited his practice to pediatrics.

AMA AHA Tighten Up On Foreign Grads

Hospitals employing graduates of foreign medical schools who fail to meet certain requirements by December 31 will face action by the American Hospital Association.

At that time, the AHA will cease listing hospitals which employ in patient-care situations foreign medical graduates who have not been certified by the Educational Council for Foreign Medical Graduates, Edwin L. Crosby, M.D., AHA director, said today.

Explanation of the ruling appears in a question-and-answer pamphlet now being mailed to all hospitals which are members of the AHA.

The AHA listing program provides a national census of hospitals that meet specific requirements. Listing is not intended to be a measure of quality or care, Doctor Crosby said. However, listing is a prerequisite for accreditation by the Joint Commission on Accreditation; thus if a hospital loses its listing, it automatically loses its accreditation.

The AHA ruling follows that of the American Medical Association, which has stated that it will withdraw approval of internships and residencies in hospital employing foreign graduates after July 1, unless the appointees meet one of the following requirements:

Possesses a full and unrestricted state license to practice; is in his final six months of training; possesses full or temporary certification from the Educational Council; or has been given a six-month appointment contingent on passing the September 21, 1960, examination by the Educational Council.

The Council was organized in 1957 for evaluating the qualifications of graduates of foreign medical schools. It is sponsored by the AHA, AMA, the Association of American Medical Colleges, and the Federation of State Medical Boards of the United States. The examination includes verification of educational credentials; an oral English test, and a written test of medical knowledge.

The Council was formed because of the number of graduates coming to the U.S. for advanced medical training and the difficulty in maintaining adequate information about foreign schools. Foreign graduates who are not in situations where they give direct care to the patient are exempt from certification. Graduates of Canadian and Puerto Rican medical schools are considered to be graduates of American schools.

"M.D. U.S.A." To Be Seen May 27

Oklahoma City's WKY-TV (Channel 4) will carry Smith, Kline and French Laboratories' latest program in the March of Medicine Series on Friday, May 27, from 9:00-10:00 p.m. Entitled "M.D. USA," the program is a "spectacular" in that camera crews were sent hopscotching over the face of America to film the story.

Traveling in jet airliner and on dogsled, the crews made on-the-spot reports from an isolated Alaskan village, from the delivery room of a small Louisiana hospital, from the great Navajo reservation in Arizona, from the office of a suburban Philadelphia physician, and from the rugged Door Peninsula of Wisconsin.

The story told is a simple but important one: Wherever Americans live, however they talk, dress or look, a doctor is on hand with skill, sympathy and understanding. And he is there because of dedication, the degree of which has not lessened through the years.

Members of the medical profession are encouraged to watch this excellent public relations presentation, and to invite their patients to do likewise.

ATTEND

THE OKLAHOMA WHITE HOUSE CONFERENCE ON AGING

JUNE 7 • 8 • 9, 1960

NORMAN, OKLAHOMA

Armed Forces Will Call Deferred Physicians

The Armed Forces will continue to require the services of most physicians liable for military service under the Universal Military Training and Service Act.

Lt. General Lewis B. Hershey, Director of Selective Service, issued this reminder to physicians when it became apparent recently that the Armed Forces would not call to active duty a small number of physicians in a few specialties who had been deferred for residency training under the Armed Forces Reserve Medical Officer Commissioning and Residency Consideration Program.

All reserve officers deferred for residency in most specialties will be called.

Shortages exist and will continue in certain specialties and in the group of officers who have not specialized, according to information received by the Director of Selective Service from the office of Doctor Frank B. Berry, Assistant Secretary of Defense (Health and Medical).

The Selective Service Director urged physicians not to draw erroneous conclusions concerning the need of the Armed Forces for their services. If a substantial number of physicians, basing their decision on knowledge that a few reserve medical officers in a few specialties are not being called to active duty after residency, conclude they are not needed, existing shortages in the Armed Forces will be aggravated.

The Department of Defense has found it unnecessary to requisition physicians through the Selective Service System since early in 1957. This has been so only because sufficient numbers of physicians sought reserve commissions and thus made themselves available for call to active duty.

There is a continuing need for applications for the residency program, as well as for reserve commissions and active duty at the conclusion of internship, General Hershey stressed.

The temporary surplus in some specialties in the residency program is understandable. Estimates of needs must be made four or five years ahead. Other factors are revisions in Armed Forces strength, redistribution of troops, reorganization of the hospital system, specialists choosing a military career, and voluntary extension of duty tours by reserve officers.

Nuclear Society Will Meet in Colorado

The Seventh Annual Meeting of The Society of Nuclear Medicine will be held at the Stanley Hotel, Estes Park, Colorado, June 22-25, 1960. Henry H. Turner, M.D., Oklahoma City, is President of the group.

Over seventy speakers will present new scientific information covering every phase of research, medicine and surgery as it concerns the use of nuclear phenomena in the diagnosis and treatment of disease. Special emphasis will be given to the diagnosis and treatment of thyroid disease, therapeutic use of radioisotopes and "tools of the trade."

The First Annual Address of the Nuclear Pioneers Series will be delivered by Dr. Edward Teller of the University of California. This first address will be in honor of the late Doctor Ernest O. Lawrence.

The meeting is open to all physicians, veterinarians, nurses, physicists, technicians and other scientists working with, or interested in utilizing radioisotopes in the health field. There is a nonmember registration fee of $5.00. Registration forms and copies of the program may be secured by writing to Mr. Samuel N. Turiel, Administrator, The Society of Nuclear Medicine, 430 N. Michigan Avenue, Chicago 11, Illinois.

An Apology To Wesson Oil

The advertising index of the April issue credited the ad of the Wesson Oil and Snowdrift Sales Company to another advertiser.

Wesson Oil is a valued contributor to the *Journal* and the Editorial Staff wishes to take this opportunity to correct an unfortunate error.

Coming Meetings

UNIVERSITY OF OKLAHOMA MEDICAL CENTER
POSTGRADUATE PROGRAM*
Individual Postgraduate Courses

CARCINOMA OF THE SKIN—May 27-28. Surgery, Radiology, Pathology Conference.

SERIAL POSTGRADUATE COURSE
Postgraduate Division
Oklahoma City, Oklahoma
1959-1960

Wednesday Short Courses
3:30 to 8:30 p.m.

June 8 Practical Considerations of Neurological Problems

*Further information may be obtained from the Office of Postgraduate Education, University of Oklahoma School of Medicine, 801 N.E. 13th Street, Oklahoma City, Oklahoma.

POSTGRADUATE COURSE IN PEDIATRICS
The University of Colorado School of Medicine
September 1-6, 1960 **The Stanley Hotel**
Estes Park, Colorado

The Department of Pediatrics and the Office of Postgraduate Medical Education, University of Colorado School of Medicine will present a Course in Pediatrics at the Stanley Hotel, Estes Park, Colorado, September 1-6, 1960.

ROCKY MOUNTAIN CANCER CONFERENCE
July 20, 21, 1960 **Denver Hilton Hotel**
Denver, Colorado

The 14th Annual Rocky Mountain Cancer Conference will be held at the Denver Hilton Hotel in Denver, July 20, 21, 1960. Details may be obtained from the Rocky Mountain Cancer Conference, 835 Republic Building, Denver 2, Colorado.

SECOND ANNUAL
OREGON CANCER CONFERENCE
July 7-8, 1960 **Portland, Oregon**

The second Annual Oregon Cancer Conference will be held in Portland, July 7-8, 1960. A copy of the complete program may be obtained by writing to Roscoe K. Miller, Executive Secretary, Oregon State Medical Society, 2164 S. W. Park Place, Portland 5, Oregon.

6th INTERNATIONAL CONGRESS OF
INTERNAL MEDICINE
August 24-27, 1960 **Basle, Switzerland**

The 6th International Congress of Internal Medicine will convene in Basle, Switzerland, August 24-27, 1960. Simultaneous translation for the principal speakers will be given in German, French and English. Programs and registration forms are available from the 6th International Congress of Internal Medicine, Steinentorstrasse 13, Basle 10, Switzerland.

Deaths

Allison T. Graham, M.D.

1891-1960

Allison T. Graham, M.D., an Oklahoma City physician since 1920, died March 28, 1960.

A native of Eastman, Mississippi, Doctor Graham graduated from Memphis Hospital Medical College of Tennessee in 1913. He served two years as a major in the Army Medical Corps during World War I.

An Honorary-Life Membership in the Oklahoma State Medical Association for Doctor Graham was pending at the time of his death.

James Harold Parker, M.D.

1886-1960

James Harold Parker, M.D., retired Oklahoma City psychiatrist and veteran of 26 years' service with the federal government, died April 21, 1960 in Oklahoma City.

A native of Berryman, Missouri, Doctor Parker graduated from the University of Louisville School of Medicine in 1910. Before joining the Veterans Administration, Doctor Parker served as superintendent at state mental hospitals in Farmington and St. Joseph, Missouri.

His career with the Veterans Administration began in 1930 at Alexandria, Louisiana. He later served at Veterans Hospitals in Waco, Texas, Sheridan, Wyoming and Muskogee, Oklahoma before coming to Oklahoma City.

Auxiliary News

Finale

Our Oklahoma State Convention held May 1 to 4 in Oklahoma City provided an enthusiastic recapitulation of the past year's activities, plus a forward look to the coming year under the direction of new officers. We sincerely thank Mrs. Clifford M. Bassett, past State President, and all of her coworkers. We warmly welcome our new President, Mrs. Virgil Ray Forester, our new officers and chairmen, as they initiate plans for the coming year.

On to A.M.A.

As our own convention concludes this phase of our county activities, so the 1960 Woman's Auxiliary to the American Medical Association climaxes the composite work of all of our state auxiliaries. This year the convention will be held June 13 to 16 at Miami Beach, Florida.

Hotel Deauville will be headquarters for the pre-convention meetings which start June 11. Oklahoma will be represented by Mrs. Bassett and Mrs. Forester at these meetings. Mrs. Neil Woodward, National Civil Defense Chairman, will present a skit, "Operation Home Preparedness" during the morning session of the regular convention, June 14.

The following day Mrs. Frank Gastineau, out-going National President, will present the gavel to our new President, Mrs. William Mackersie. Mrs. Richard F. Stover of Miami, Chairman for the 37th Annual Convention, invites all members to participate in a stream-lined convention program, with ample free time allowed for sight-seeing and shopping.

Bring Your Family

From the advance bulletin: "Cabana parties, sight-seeing cruises—even ice-skating —are included in the entertainment planned for elementary and teen-age children attending the AMA convention with their parents. The Deauville boasts the only artificial ice-skating rink on the famous Gold Coast. Entertainment for the young people will get under way Monday, June 13, with a boat ride which takes them through the beautiful man-made islands. Two trips—one for the elementary group, one for the teen-age—will be made. A pool and patio supper party takes place Monday evening from 6 to 10:30 for the teen-agers, on the beach and around the Deauville cabanas."

Tours of Miami, Miami Beach and points of interest in Dade County will be available on Tuesday, Mrs. Stover announces. She adds that "children of elementary school age will have supervision, so that if mother is in a committee meeting, she won't be worrying about her child."

Lyla Dix, Publicity Chairman for the Convention, has helpfully issued the following information: Miami in June promises highs of 86 degrees and minimums of 77 degrees, emphasizing a choice of packing: Come with empty bags and take advantage of the sales of resort clothes. Or, if you prefer to "bring your own," keep in mind that Miami Beach is "casual and sophisticated, but not careless." Shorts, slacks and beach attire are the order of the day and may be worn, in addition to the beach and sports events, in the lower lobby and the coffee shop of the Deauville; not in the main dining room or the main lobby. Linens, cottons and comfortable summer things are stressed. Jackets and sweaters are musts for air-conditioned meeting rooms.

Our general invitation to Miami Beach and AMA is: Come—Bring the Family—and Have a Wonderful Time!

25 YEARS AGO



Articles published in *The Journal* of the Oklahoma State Medical Association, May, 1935.

THE USE OF DILAUDID IN GENERAL PRACTICE

Clifford M. Bassett, M.D., Cushing

The drug dilaudid is a morphine derivative, dihydromorphine hydrochloride, a rearrangement of the morphine molecule. This change in chemical structure of the morphine molecule increases the analgesic effect about five times, appears to remove the constipating effect and also seems to eliminate much of the euphoric effect of morphine. My observation has shown that the nauseating and emitic effect of morphine are almost entirely absent. With one or two patients I observed slight dizziness and confusion, but this was transient, lasting but a few minutes.

Dilaudid, in general practice, gives the physician a drug that can be employed in many varied conditions to bring relief from pain. It is a powerful analgesic, with a prompt and definite action. Following a specific dose, a corresponding certain degree of analgesia may be expected. I have given patients morphine in ¼ grain doses only to find it necessary to repeat the dose in 30 to 40 minutes; with dilaudid I have not encountered a case that either 1/32 or 1/20 of a grain did not give complete and persistent relief. I have not found it necessary to give large doses, as the effect of dilaudid persisted for some time following its administration. In the post-operative cases many of the patients go from six to eight hours before requiring further analgesic medication.

The relaxing properties are also apparent in the post-operative period as shown by the infrequent occurrence of urination retention, requiring catheterization. It was not necessary to catheterize during the post-operative course when dilaudid was used for pain. Those patients for which catheterization was advised for a time following the operation, as for example, after perineorrhaphy, were able to void easily when allowed to do so. No doubt in a larger series it might be necessary to occasionally catheterize a patient post-operatively but if the necessity for this procedure is reduced to a minimum, that factor alone should invite the universal adoption of dilaudid over any other opiate in post-operative pain.

I have also used dilaudid as a cough sedative with good results. It exhibits a sedative effect similar to heroin and in very small doses. I have used as little as 1/20 to 1/10 grain in one ounce of vehicle. It does not inhibit secretions, induce constipation, nor do the patients complain of drowsiness.

One factor which makes the drug pleasant to work with is its solubility. The time consumed in preparing hypodermic medication is greatly reduced, and this, plus rapidity of action of the drug, insures relief to the suffering patients in the shortest possible time.

Have You Heard?

PAUL KERNEK, M.D., Holdenville, was recently appointed by Governor J. Howard Edmondson to fill an unexpired term on the State Board of Health. The vacancy occurred when M. L. Whitney, M.D., Okemah, resigned.

W. D. ANDERSON, M.D., M. E. GORDON, M.D., O. U. HOLT, M.D., M. R. JENNINGS, M.D., and ROBERT M. STOVER, M.D., of Claremore, have set up a scholarship for a student nurse each year for the next three years. The girls selected by the physicians may attend any accredited nurses' training school. Following graduation, the nurses will be expected to spend at least one year at the Claremore Health Center, providing there is an opening at that time.

PHILLIP SHEPPARD, M.D., Tulsa, will be associated with Eugene Bell, M.D., in the Bell Clinic in Tishomingo after July 1. Doctor Sheppard graduated from the University of Oklahoma School of Medicine before taking his internship at St. John's Hospital in Tulsa.

RALPH BULLER, M.D., Lafayette, Louisiana, will establish his practice in Hydro after September 15. Doctor Buller, a graduate of the University of Oklahoma School of Medicine in 1956, served two years in the Air Force before taking a residency at the Charity Hospital of Louisiana.

PHYSICIAN PLACEMENT

Dermatology

Gordon Harold Ekblad, M.D., Field Medical Service School, Camp Pendleton, California, 53, married, University of Minnesota, 1930, available January 1960.

Lealand L. Clark, M.D., 200 1st St., S.W., Rochester, Minnesota; age 28; married; Columbia University College Physicians and Surgeons 1956; Board eligible July 1960; Fellows' Assoc., Mayo Foundation; Military service will be completed Sept. 1962; desires group or associate practice; available July 1960.

Joseph William Pidgeon, M.D., 5333 Littlebow Road, Rolling Hills, California; age 41; married; George Washington University 1951; Board eligible June 1960; Active Air Force Reserves; available July 1960.

General Practice

Johnny Bill Delashaw, U.S.P.H.S. Hospital, Wyman Park Drive and 31st Street, Baltimore 11, Maryland, age 27, married, University of Texas Medical Branch, 1959, available July 1960.

Robert G. Bissell, M.D., 143 S. Wheeling, Tulsa, Oklahoma, age 28. married; University of Oklahoma School of Medicine, 1959; no military obligations; available July 1, 1960.

Robert Goodman, M.D., 63 Old Gate Lane, Powers Lake, North Dakota; age 56; married; University of Manitoba, 1930; no military obligations; available anytime 1960.

Richard Fred Harper, M.D., Conway Memorial Hospital, Monroe, Louisiana; age 28, married; Oklahoma University, 1956; available July 1960; prefer associate practice.

Sherman Allen Hope, M.D., 1105 Yale Ave., Panama City, Florida, PO 3-6656, age 27, married, University of Oklahoma School of Medicine, 1957, available July 1960.

Internal Medicine

Richard I. Hochman, M.D., 37, Badger Road, Annapolis, Maryland, 31, married, New York University, 1952, available October 1, 1960.

Edward John Hertko, M.D., 2117 S. 3rd Ave., Maywood, Illinois; age 30; married; University of Illinois, 1954; no military obligations; available October 1960.

Joseph Chester Maternowski, M.D., 319 Highlake, Ann Arbor, Michigan; age 35; married; St. Louis University, 1953; eligible board of Internal Medicine & Allergy; veteran; specialty, Internal Medicine & Allergy; available July 1, 1960.

Locum Tenens

Harold W. Calhoon, M.D., Wesley Hospital, Oklahoma City. One year residency in Internal Medicine. Available July 1 to September 15th, 1960.

Arnold Giesbrecht, M.D., Hallock, Minnesota, age 34, married, graduate Winnipeg, Manitoba, 1957, prefers suburban or locality close to cities.

James D. Green, M.D., second year resident in Internal Medicine at St. John's Hospital, Tulsa, desires two weeks' tenure between now and July 1. Write to St. John's or call RIverside 3-3059.

(Name on Request), O.U. graduate 1956; M.D. from Washington University School of Medicine 1959; desires locum tenen in the Tulsa area from July 1, 1960 until middle of September, 1960. Address inquiries to Key H, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

Charles L. Maimbourg, M.D., Campus Apartments No. 5, Elf Street, Durham, North Carolina; Oklahoma University School of Medicine 1958; desires General Practice and/or Anesthesia during first two or three weeks of July 1960.

Russell M. Preston, M.D., 476 San Juan, Oak Harbor, Washington, desires six month's work in General Practice between time of separation from military service to beginning of residency training, January-July, 1960.

Obstetrics and Gynecology

Edmond Michael Brophy, M.D., 1823 Portsmouth, Westchester Illinois; married; University of Illinois, 1946; Board eligible upon completion of practice requirements; no military obligations; available July, 1960.

William Edgar Carlisle, M.D., 634 E. Patton Ave., Montgomery, Alabama; age 29; married; Tulane University School of Medicine 1953; Jr. Fellow American College of Ob-Gyn; interested in associate or group practice; available July 1960.

Robert Russell Fahringer, M.D., P.O. Box 148, Danville, Pennsylvania; age 35; married; Jefferson Medical College of Philadelphia, 1953; inactive military status, USNR; prefers full or part-time teaching; available February 1960.

Joseph William Herbert, M.D., 2701 N. Azelea St., Victoria, Texas; age 40; married; Southwestern Medical School, 1945; Fellow American College of Ob-Gyn; desires associate or solo practice; no military obligations; available March 1960.

David H. Holmes, M.D., 1017 Grovena Drive, St. Louis, Missouri, 37, married, Washington University, St. Louis, Veteran, available July 1, 1960.

Gerald R. Keilson, M.D., Medical Arts Building, Dallas, Texas, age 31, married, University of Texas, 1953, board qualified, veteran, available in July 1960.

Nejdat Mulla, M.D., 1110 Belmont Avenue, Youngstown, Ohio, age 36, married, University of Geneva, Switzerland, 1952, American College of OB-GYN, not eligible military service, available July 1, 1960.

Oklahoma physician; age 35; married; Medical College of Georgia, 1954; 3 years residency training Ob-Gyn; veteran; available in 30 days. Write Key "I", Oklahoma State Medical Association, P.O. Box 9696, Shartel Station, Oklahoma City, Oklahoma.

Harold G. Ray, M.D., 212 Darien Place, Box 503, Balboa Heights, Canal Zone, age 28, married, University Arkansas School of Medicine, 1955, now in active reserve, available July 1, 1960.

Harold E. Reid, M.D., 519 N. Lincoln St., Stanton, Michigan; age 42; married; University of Michigan, 1942; OB-GYN, also interested in industrial; available February 1960.

Juan C. Ruiz, M.D., 350 Park Ave., Lexington, Kentucky; age 36; married; University of San Marcos Medical School 1952; no military obligations; available July 1, 1960.

Pediatrics

H. M. McClintock, M.D., 5712 St. John St., Kansas City, Missouri, 29, married, Baylor University, 1955, veteran, available December 1960.

Ophthalmology

Howard A. Dinsdale, M.D., 4624 Briarfield Road, Columbia, South Carolina; age 29; married; University of Nebraska 1954; Board Certified; available October 11, 1960.

Ira H. Kaufman, 75 S. Middle Neck Road, Great Neck, New York; age 31; married; Cornell University 1953; Board certified; desires group or associate practice; available summer 1960.

Orthopedics

Newsom Stool, M.D., c/o May Clinic, Rochester, Minnesota; age 30; married; Baylor University 1953; Board eligible; available July 1960.

Surgery

(Name on Request) 32 years old, married, Tulane, 1952, veteran, board eligible.

Clarence I. Britt, M.D., 1650 Neil Avenue, Apt 15, Columbus, Ohio; age 32; married; Ohio University College of Medicine, 1951; American Board of Surgery 1959; eligible American Board of Thoracic Surgery, 1960; inactive reserve; available July 1960.

Delbert H. McGinnis, M.D., Box 75, Clinton-Sherman AFB, Oklahoma; age 28; married; end military obligation July 3, 1960; interested in general practice; available July 4, 1960.

Duane M. Clement, M.D., 310 Bates, Boulder, Colorado; age 39; married; University of Michigan 1944; Board certified; member F. A. Coller Surgical Society; desires group or associate practice; available April 1960.

Alice F. Gambill, M.D., 1601 Clover Lane, Fort Worth, Texas; age 36; married; Oklahoma University School of Medicine 1949; member AAGP; desires general or group practice; available May 1960.

Donal Wray Steph, M.D., 8519 Craighill, Dallas, Texas; age 30; married; Southwestern Medical School 1953; board eligible July 1960; available July 1960.

Clinton K. Higgins, M.D., 215 Bacon Ave., Naval Base, Norfolk, Virginia; age 62; married; Washington University 1927; Board Certified; desires administration, college health physician, group association; available May 1960.

Urology

Alex Grossman, M.D., V.A. Center, Temple, Texas, 42, married, University of Texas. 1951, board certified, veteran, available now.

John A. Malley, M.D., 3404 Sheffield Ave., Los Angeles, California; age 32; married; Marquette University 1955; Board certified; no military obligations; available July 1, 1960.

MISCELLANEOUS ADVERTISEMENTS

FOR SALE: Autoclave, Castle, electric. Originally purchased for $1866.75 in 1956. Contact Walt Allen, 116 North Fourth Street, Chicaksha, Oklahoma.

FOR SALE or RENT: Office building, equipment and supplies, including x-ray. Residence also available. Contact Box F, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: Fluoroscope (1 only), G. E. Cat. #B-3002A, 100 to 130 Volts, 50/60 Cycles with 10 M.A. Transformers and Control, Vertical Roentgenoscope, including mounting for R. B. Type Tube, with 12″ x 16″ Patterson Type B Fluoroscopic Screen. May be inspected at the Department of Public Welfare, Supply Room, Capitol Building, Room No. 5, Oklahoma City, Oklahoma. This item is being sold on bid through the State Board of Public Affairs on Sale #66. Bids may be obtained from either the State Board of Public Affairs or the State Department of Public Welfare and the bids close on June 15, 1960.

FOR LEASE OR RENT: Completely equipped for general practice, 3-room clinic, 26 S.W. 25th, Oklahoma City. Seven years old. Air conditioned, central heat. Will rent with or without furniture and equipment. Contact Mrs. Zelma King, 222½ S.W. 25th, Oklahoma City. CE 2-2575 or ME 2-6811.

FOR SALE: General practice, established six years, in oil and ranching community. Town population is 3,000 with trade area over 15,000. Closest physician 30 miles. Netting $20,000 to $24,000 per year. Located in modern, leased clinic building. Presently in partnership but continued partnership not mandatory. 18-bed, modern, community hospital. Only two physicians in town. Reason for selling, physician returning to Hawaii. No down payment necessary. Contact William G. Mays, M.D., Fairfax, Oklahoma.

WANTED: Young General Practitioner, without military obligations, to become partner in established clinic and hospital practice. $1000.00 per month to start. Contact Neumann-Ottis Clinic, Okarche, Oklahoma.

DEPUTY FLIGHT SURGEON wanted for Federal Aviation Agency. The 40 hours-per-week position pays an annual salary of $12,555, plus Civil Service benefits including a retirement program, annual and sick leave policy, group life insurance and others. Physician must have completed the basic course in Aviation Medicine prescribed by the U.S. Military Service, and have three years professional experience in internal medicine. Write FAA, Region 2, Box 1689, Ft. Worth 1, Texas.

WANTED: Internist to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR RENT: Office space for two doctors, modern, air conditioned building. 3832 N. May Ave., Oklahoma City. Complete maintenance. Free parking space for patients. WI 2-5531.

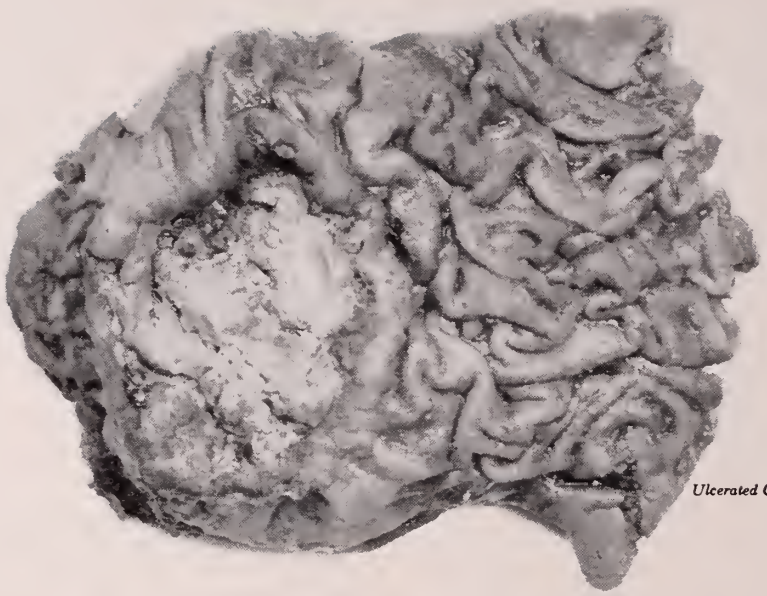
WANTED: Surgeon to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: Office with six rooms, small laboratory, two rest rooms, storage room, air conditioned. Equipped for E.E.N.T. Near hospital. Nicely finished, with cement, cork covered floors. Good location. Equipment includes specialist's chair with stand. Phoropter, projector and space eikonometer. Special trial case, giant scopes. Ritter unit, Diatherma, Two Violet Ray units, instruments, tables, chairs, etc. Contact Edward M. Loyd, M.D., P.O. Box 2061, Harlingen, Texas.

FOR SALE: General practice partnerships established 7 years in prosperous oil and agriculture community. Town of 5000 and trade area over 15,000 population. Gross receipts exceed $50,000 annually. New clinical building for sale or rent. Practice is priced reasonably and terms can be arranged. Owners going into residency. Contact Box L, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: New $3250.00 Profexray, 100 MA, 100 KV X-ray unit with: rotating anode tube and examining type table with built in recipromatic bucky and table mounted tube stand, foam rubber pad for table and heel stirrups, transformer and control with shock proof cables including 1/30th second timer, one floor mounted cassette holder, one Videx rectangular cone with center light. Price $2500.00. Can be seen at Merkle X-ray Co., Tulsa, Oklahoma. Contact E. Malcolm Stokes, M.D., 2021 South Lewis, Tulsa.

NEGRO PHYSICIAN wanted for Chickasha, Oklahoma. Contact R. C. Alexander, 917 South 2nd Street, Chickasha, Oklahoma.



Ulcerated Carcinoma of the Stomach

what's new in the treatment of cancer?

Fifteen cancer authorities discuss latest advances in treatment of malignancies in a combined symposium and panel discussion at 9 a.m., June 15, in the Miami Auditorium. This is one of three sessions the A.M.A. Section on General Surgery presents at the Annual Meeting in Miami Beach June 13—17. More than 300 scientific presentations are scheduled.

The Symposium:

Recent Advances in the Treatment of the Cancer Patient

Warren H. Cole, Chicago, President, American Cancer Society; Head, Department Surgery, University of Illinois: "*An Introduction to the Problem*".

Charles L. Eckert, Albany, N. Y., Chairman, Department of Surgery, Albany Medical College: "*Extended Surgical Procedures in Treatment of Cancer.*"

George E. Moore, Buffalo, N. Y., Director, Roswell Park Memorial Institute: "*Modification of Host Resistance to the Growth of Malignant Cells.*"

Anthony R. Curreri, Madison, Wis., Director, University Cancer Research Hospital; and by

Lyndon E. Lee, Washington, D. C., Coordinator, Vet. Adm. Program on Cancer Chemotherapy: "*Status of Adjunct Cancer Chemotherapy.*"

John D. Hurley, Milwaukee, Assistant Director of Surgery, Milwaukee County Hospital: "*Chemotherapy of Solid Cancer.*"

William R. Richardson, Oklahoma City, Professor of Pediatric Surgery, University of Oklahoma: "*Newer Techniques of Managing Neoplasms in Infants and Children.*"

Benedict R. Walske, Omaha, Neb., Chairman, Department of Surgery, Creighton University: "*Effects of Radioactive Iodinated Tetracycline on Growth of Neoplasms.*"

R. Lee Clark, Houston, Director and Surgeon-In-Chief, University of Texas: "*Local Perfusion Techniques in Chemotherapy of Inoperable Cancer—Indications, Methods and Results.*"

The Panel Discussion:

Current Status of Treatment of Advanced Cancer of Thyroid and Breast

Warren H. Cole, Chicago, Moderator
Walter T. Murphy, Buffalo, N. Y.
George Crile, Jr., Cleveland, Ohio
Joseph Farrow, New York City
Bronson S. Ray, New York City
Arthur James, Columbus Ohio
Fred Ansfield, Madison, Wis.

ANNUAL MEETING,
Miami Beach, Florida
June 13-17, 1960

Advance hotel and meeting registration forms appear in the first issue of JAMA each month, beginning in February.

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she calls it "nervous indigestion"

diagnosis: a wrought-up patient with a functional gastro-intestinal disorder compounded by inadequate digestion. **treatment:** reassurance first, then medication to relieve the gastric symptoms, calm the emotions, and enhance the digestive process. **prescription:** new Donnazyme—providing the multiple actions of widely accepted Donnatal® and Entozyme®—two tablets t.i.d., or as necessary.

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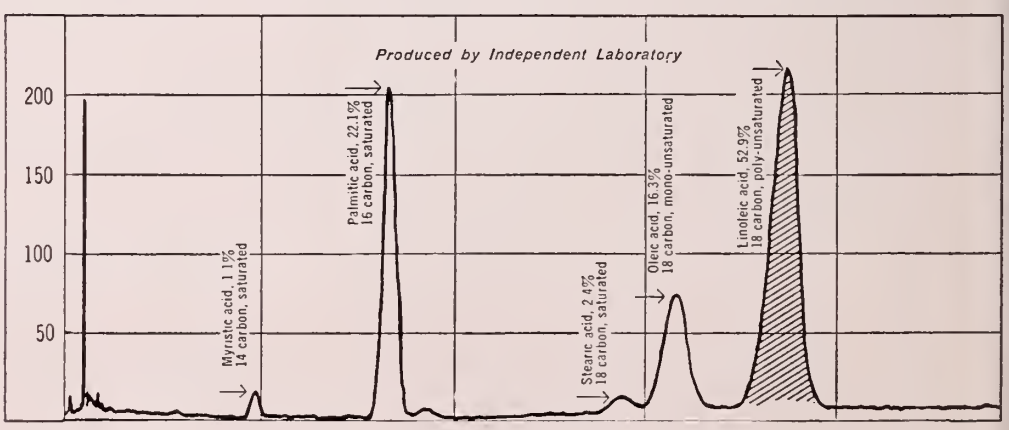
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"It is now well recognized that serum cholesterol levels in man can be lowered by the judicious substitution of one type of dietary fat for another. However, it is relevant to inquire whether a patient can be assured that such a radical change in his dietary habits will prevent coronary occlusion or a cerebral vascular accident. This question must unfortunately be answered in the negative, for it has not been proved that lowering the level of serum cholesterol will prevent either the occurrence or the end-results of atherosclerosis. At the present time, clear proof of this proposition still seems many years away. Nevertheless, there are many reasons for believing that there is some connection between cholesterol metabolism and atherosclerosis, and, while waiting for elucidation of this relationship by laboratory workers, it seems justifiable to apply certain dietary procedures that are theoretically harmless and possibly beneficial."

Excerpted from J.A.M.A., Aug. 29, 1959

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ACTS FASTER—usually within 5-15 minutes. **LASTS LONGER**—usually 6 hours or more. **MORE THOROUGH RELIEF**—permits uninterrupted sleep through the night. **RARELY CONSTIPATES**—excellent for chronic or bedridden patients.

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in infectious disease[17,22,30,36]
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in peptic ulcer[8,21,38]
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11 vitamins, 8 minerals clinically-formulated and potency protected to provide enough nutritional support to do some good

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1-41 a list of the above references will be supplied on request.

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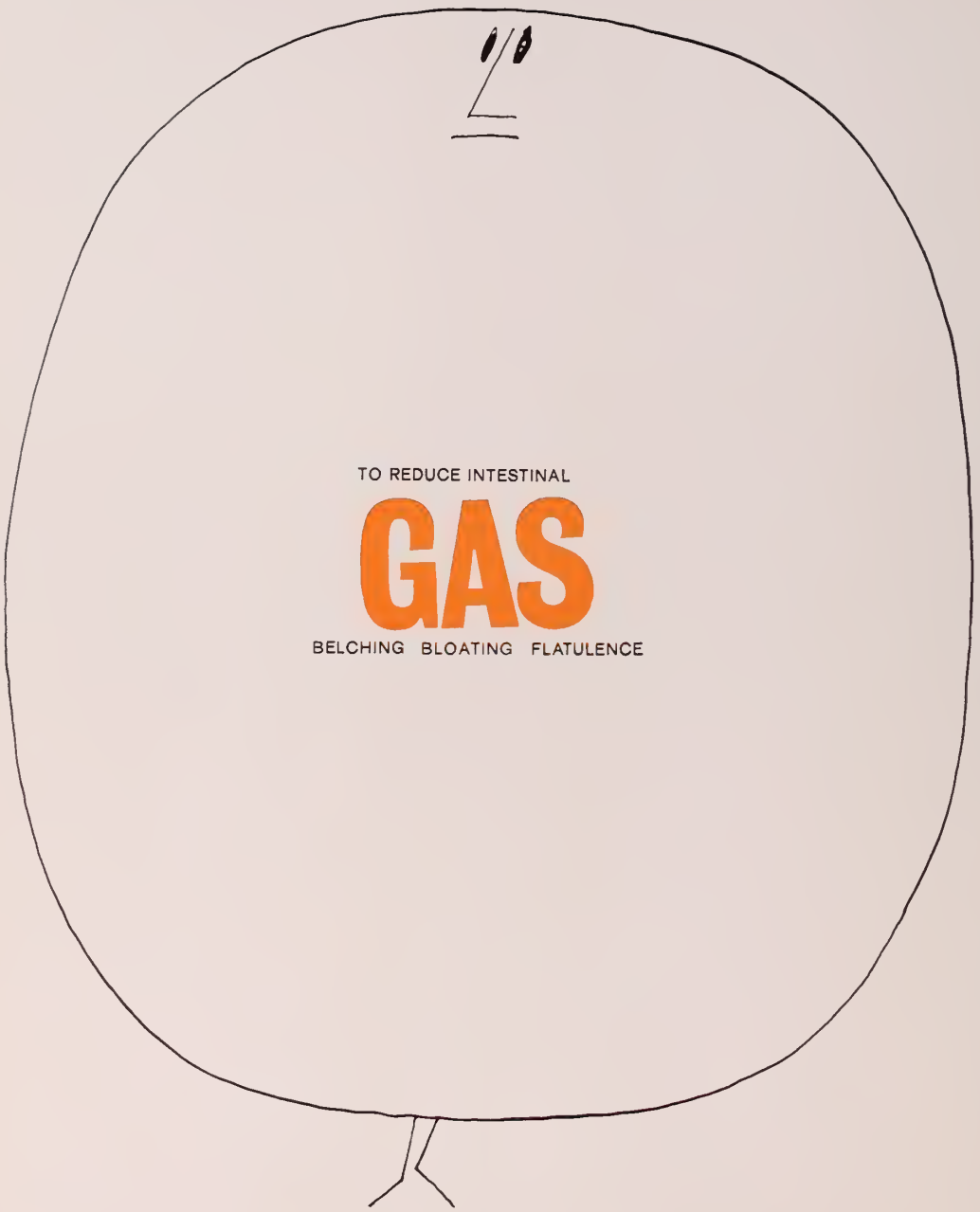
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in depression

lights the road to recovery in 80 per cent of cases



In the treatment of depression Tofrānil has established the remarkable record of producing remission or improvement in approximately 80 per cent of cases.[1-7]

Tofrānil is well tolerated in usage—is adaptable to either office or hospital practice—is administrable by either oral or intramuscular routes.

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a potent thymoleptic . . .
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Does act effectively in *all* types of depression regardless of severity or chronicity.

Does not inhibit monoamine oxidase in brain or liver; produce CNS stimulation; or potentiate other drugs such as barbiturates and alcohol.

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References: 1. Ayd, F. J., Jr.: Bull. School Med., Univ. Maryland *44*:29, 1959. 2. Azima, H., and Vispo, R. H.: A.M.A. Arch. Neurol. & Psychiat. *81*:658, 1959. 3. Lehmann, H. E.; Cahn, C. H., and de Verteuil, R. L.: Canad. Psychiat. A. J. *3*:155, 1958. 4. Mann, A. M. and MacPherson, A. S.: Canad. Psychiat. A. J. *4*:38, 1959. 5. Sloane, R. B.; Habib, A., and Batt, U. E.: Canad. M.A.J. *80*:540, 1959. 6. Straker, M.: Canad. M.A.J. *80*:546, 1959. 7. Strauss, H.: New York J. Med. *59*:2906, 1959.

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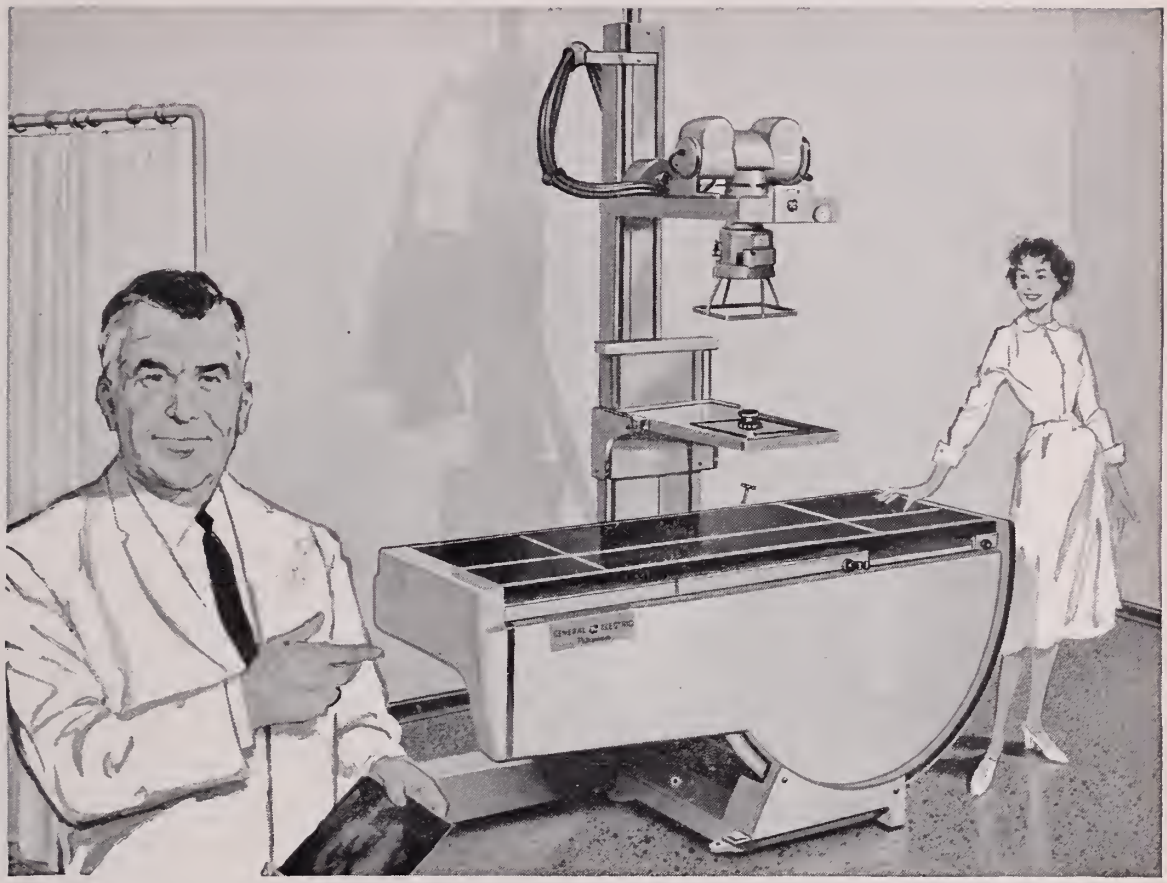
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When she asks "Doctor, what will it be?" you can either flip a coin or point out that 51.25% births are male.[1] But when she mentions morning sickness, your course is clear: BONADOXIN.

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1. Projection from Vital Statistics, U.S. Government Dept. HEW, Vol. 48, No. 14, 1958, p. 398.
2. Modell, W.: Drugs of Choice 1958-1959, St. Louis, C. V. Mosby Company, 1958, p. 347.

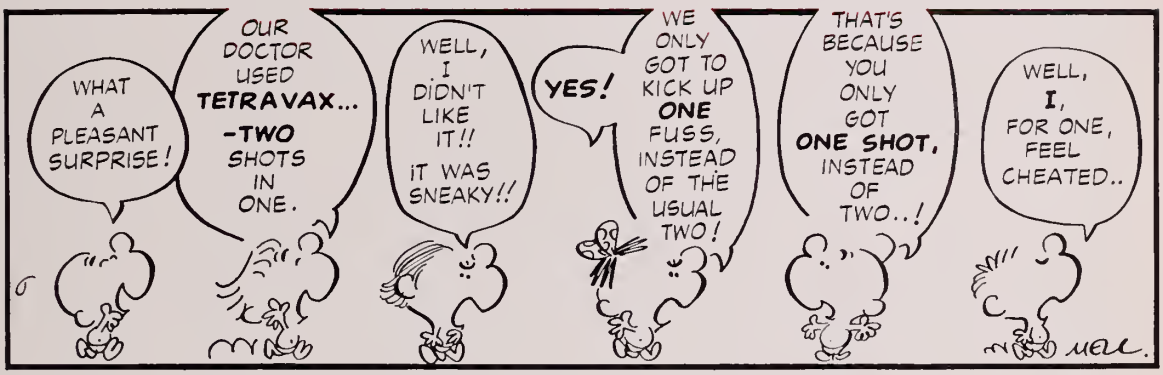
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now you can immunize against more *diseases... with* fewer *injections*

Dose: 1 cc.
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OUTSTANDING 1-DOSE-A-DAY SULFA—*Rapid peak attainment* in 1 to 2 hours[1,2]... approximately one-half the time of other single-daily dose sulfas.[2] *High free levels*—as much as 95 per cent of circulating levels remaining in fully active unconjugated forms.[3] *Extremely low 2.7 per cent incidence of side effects* in a clinical study on 223 patients.[4] Includes *total* reactions (subjective and objective), all temporary and rapidly reversed. No crystalluria reported.

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New for acute G. U. infection AZO KYNEX Tablets (for q. i. d. dosage), 125 mg. KYNEX sulfamethoxypyridazine in the shell with 150 mg. phenylazodiaminopyridine HCl in the core.

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1. Boger, W. P.; Strickland, C. S., and Gylfe, J. M.: Antibiotic Med. & Clin. Ther. 3:378 (Nov.) 1956. 2. Boger, W. P.: In: Antibiotic Annual 1958-1959, Medical Encyclopedia, Inc., New York. 1959, p. 48. 3. Sheth, U. K.; Kulkarni, B. S., and Kamath, P. G.: Antibiotic Med. & Clin. Ther. 5:604 (Oct.) 1958. 4. Anderson, P. C., and Wissinger, H. A.: U. S. Armed Forces M. J. 10:1051 (Sept.) 1959.

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brand of phenylbutazone

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proved by a decade of experience

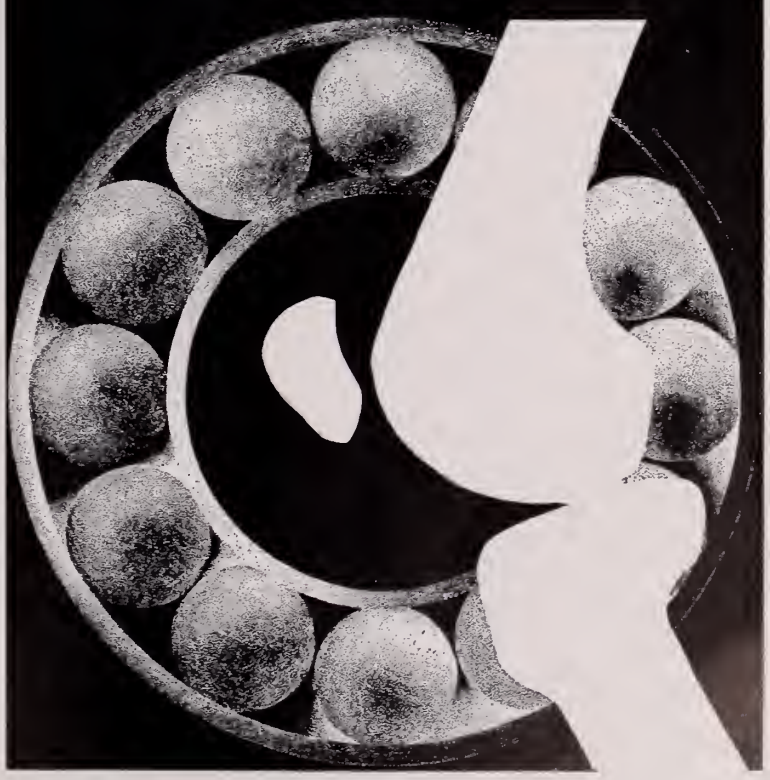
Ten years of experience in countless cases—more than 1700 published reports—have now established the eminence of Butazolidin among the potent non-hormonal antiarthritic agents.

Repeatedly it has been demonstrated that Butazolidin:
Within 24 to 72 hours produces striking relief of pain.
Within 5 to 10 days affords a marked improvement in mobility and a significant subsidence of inflammation with reduction of swelling and absorption of effusion.

Even when administered over months or years Butazolidin does not provoke tolerance nor produce signs of hormonal imbalance.

Butazolidin® brand of phenylbutazone: Red-coated tablets of 100 mg.
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more complete than antihistamines alone... more thorough than nose drops or sprays

The miseries of respiratory allergy can be relieved so effectively with Triaminic.[1-5] Triaminic contains two antihistamines plus the decongestant, phenylpropanolamine, to help shrink the engorged capillaries, reduce congestion and bring relief from rhinorrhea and sinusitis.[1] Oral administration distributes medication to *all* respiratory membranes without risk of "nose drop addiction" or rebound congestion.[2,3]

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Phenylpropanolamine HCl	50 mg.
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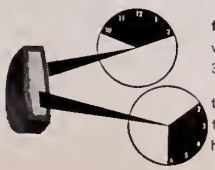
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TRIAMINIC JUVELETS® ½ the formulation of the Triaminic Tablet with timed-release action.

TRIAMINIC SYRUP each teaspoonful (5 ml.) provides ¼ the formulation of the Triaminic Tablet.

References: 1. Fabricant, N. D.: E. E. N. T. Monthly **37**:460 (July) 1958. 2. Lhotka, F. M.: Illinois M. J. 112:259 (Dec.) 1957. 3. Farmer, D. F.: Clin. Med. 5:1183 (Sept.) 1958. 4. Fuchs, M.; Bodi, T.; Mallen, S. R.; Hernando, L., and Moyer, J. H.: Antibiotic Med. & Clin. Ther. 7:37 (Jan.) 1960. 5. Halpern, S. R., and Rabinowitz, H.: Ann. Allergy **18** 36 (Jan.) 1960.

Relief is prompt and prolonged because of this special timed-release action

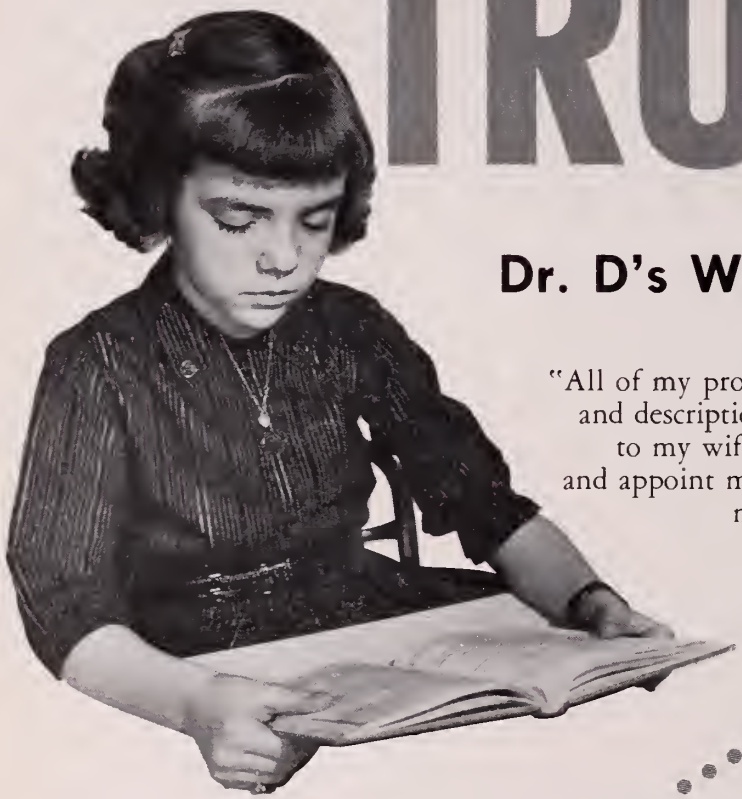


first—the outer layer dissolves within minutes to produce 3 to 4 hours of relief

then—the core disintegrates to give 3 to 4 more hours of relief

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Dr. D's Will said this . . .

"All of my property of every kind, character and description I give, devise and bequeath to my wife, Jane. I nominate, constitute and appoint my wife, Jane, executrix of this my Last Will and Testament."

But this is what it really meant:

ITEM 1.

To my wife, Jane, I bequeath all of my property, together with the risks and responsibilities of its management. I bequeath her worry for the remainder of her life. I bequeath her financial insecurity, although my estate is ample to care for her.

ITEM 2.

To my daughter, Mary, I bequeath a Mother who will have many added worries and responsibilities. To my daughter, I further bequeath an estate shrunken by unnecessary taxation.

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The reasons are fairly simple. Doctors like "Premarin," in the first place, because it really relieves the symptoms of the menopause. It doesn't just mask them — it replaces what the patient lacks — natural estrogen. Furthermore, if the patient is suffering from headache, insomnia, and arthritic-like symptoms due to estrogen deficiency, "Premarin" takes care of that, too.

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Some strains of staphylococci resistant to other penicillins exhibit in vitro sensitivity to potassium phenethicillin.

ALPEN has greater freedom from the G. I. sequelae (overgrowth of resistant flora) sometimes observed with broad spectrum -mycins.

ALPEN gives much higher antibiotic levels within the first hour of ingestion by the well-tolerated oral route.

WHEN TO USE ALPEN Recommended in the treatment of infections caused by pneumococci, streptococci, gonococci, corynebacteria, and penicillin-sensitive staphylococci.

HOW TO USE ALPEN Depending on the severity of the infection, 125 mg. (200,000 units) or 250 mg. (400,000 units) three times daily may be used. In more severe or stubborn infections, a dosage of 500 mg. (800,000 units) t.i.d. may be employed. In beta hemolytic streptococcal infections, treatment should be continued for at least ten days.

PRECAUTIONS The usual precautions in the administration of oral penicillin should be observed. For further details see package literature.

Tablets: 125 mg. and 250 mg., bottles of 25 and 100. Powder for Oral Solution (lemon-lime flavored), 1.5 Gm. bottle (125 mg. per 5 cc. teaspoonful).

this is the tablet that gives higher peak antibiotic blood levels

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ALPEN™—potassium phenethicillin

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Widest latitude of safety and flexibility—no serious adverse clinical reaction ever documented.
Chemically distinct among tranquilizers—not a phenothiazine or a meprobamate.
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IN ELDERLY PATIENTS well tolerated by debilitated patients	"...seems to be the agent of choice in patients suffering from removal disorientation, confusion, conversion hysteria and other psychoneurotic conditions occurring in old age." Smigel, J. O., et al.: J. Am. Geriatrics Soc. **7**:61 (Jan.) 1959.	Settel, E.: Am. Pract. & Digest Treat. **8**:1584 (Oct.) 1957. Negri, F.: Minerva med. **48**:607 (Feb. 21) 1957. Shalowitz, M.: Geriatrics **11**:312 (July) 1956.
IN ALLERGIC PATIENTS useful adjunctive therapy for asthma and dermatosis; particularly effective in urticaria	"All [asthmatic] patients reported greater calmness and were able to rest and sleep better...and led a more normal life....In chronic and acute urticaria, however, hydroxyzine was effective as the sole medicament." Santos, I. M., and Unger, L.: Presented at 14th Annual Congress, American College of Allergists, Atlantic City, New Jersey, April 23-25, 1958.	Eisenberg, B. C.: J.A.M.A. **169**:14 (Jan. 3) 1959. Coirault, R., et al.: Presse méd. **64**:2239 (Dec. 26) 1956. Robinson, H. M., Jr., et al.: South. M. J. **50**:1282 (Oct.) 1957.
IN HYPEREMOTIVE AOULTS does not impair mental acuity	"...especially well-suited for ambulatory neurotics who must work, drive a car, or operate machinery." Ayd, F. J., Jr.: New York J. Med. **57**:1742 (May 15) 1957.	Garber, R. C., Jr.: J. Florida M. A. **45**:549 (Nov.) 1958. Menger, H. C.: New York J. Med. **58**:1684 (May 15) 1958. Farah, L.: Internat. Rec. Med. **169**:379 (June) 1956.

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1. Kestler, O.: In The Pharmacology and Clinical Usefulness of Carisoprodol, Wayne State University Press, Detroit, 1959. 2. Berger, F. M.; Kletzkin, M.; Ludwig, B. J.; Margolin, S., and Powell, L. S.: J. Pharm. Exp. Ther. *127*:66 (Sept.) 1959. 3. Spears, C. E. and Phelps, W. M.: Arch. Pediat. *76*:287 (July) 1959. 4. Phelps, W. M.: Arch. Pediat. *76*:243 (June) 1959. 5. Friedman, A. P.; Frankel, K., and Fransway, R. L.: Papers presented at Scientific Meeting, New York State Society of Industrial Medicine, Inc., New York, Sept. 30, 1959. 6. Kuge, T.: Unpublished reports. 7. Ostrowski, J. P.: Orthopedics *2*:7 (Jan.) 1960.

Literature and samples on request

Also available on request: The Pharmacology and Clinical Usefulness of Carisoprodol, Wayne State University Press, Detroit, 1959. (185 pages)

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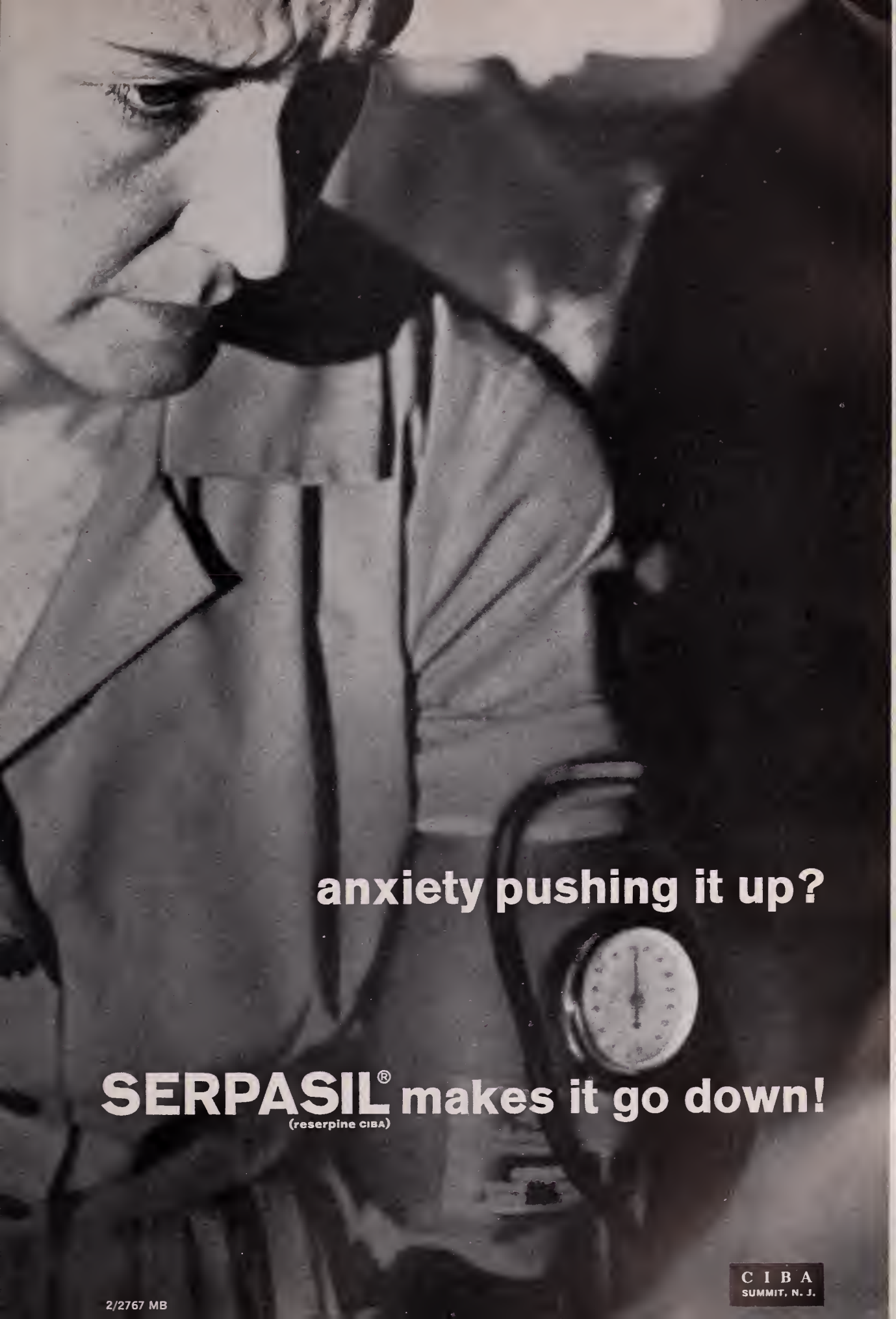


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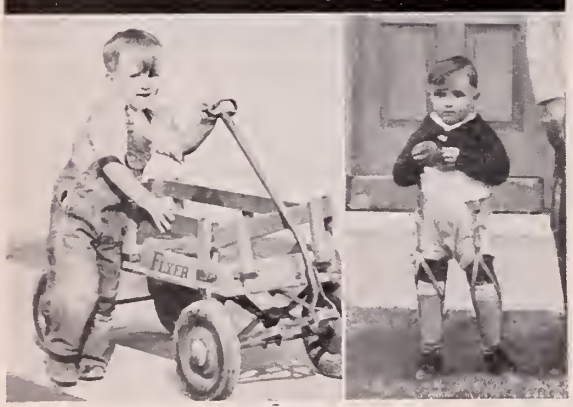
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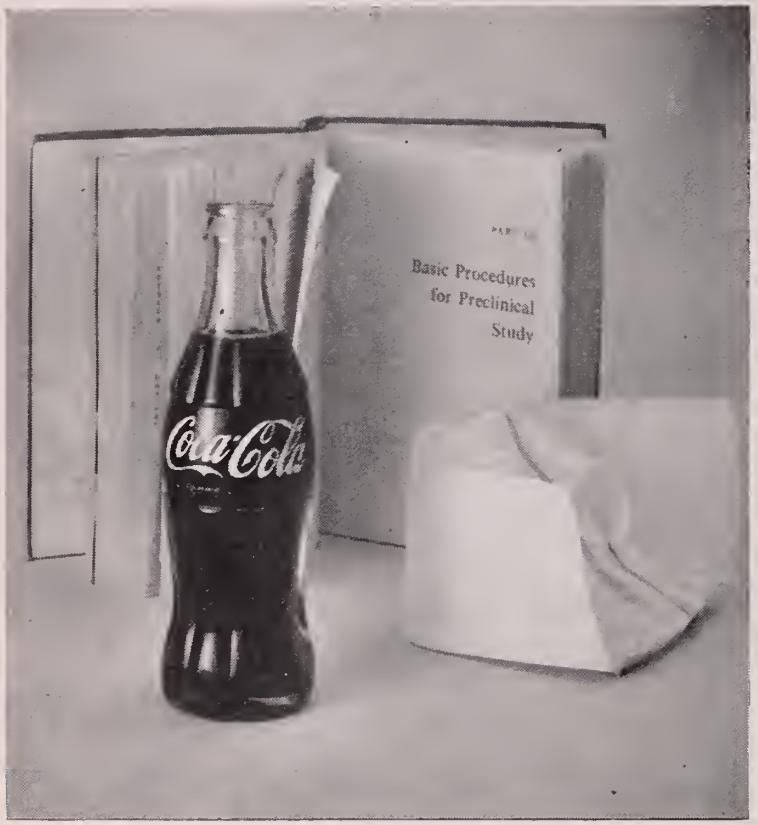
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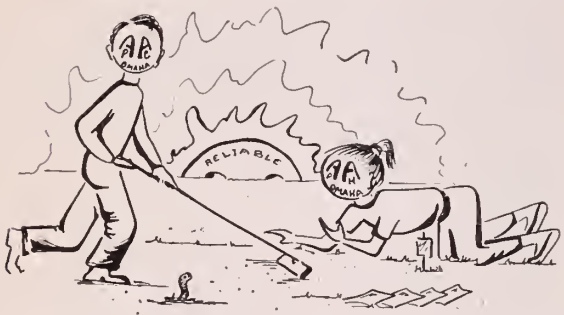
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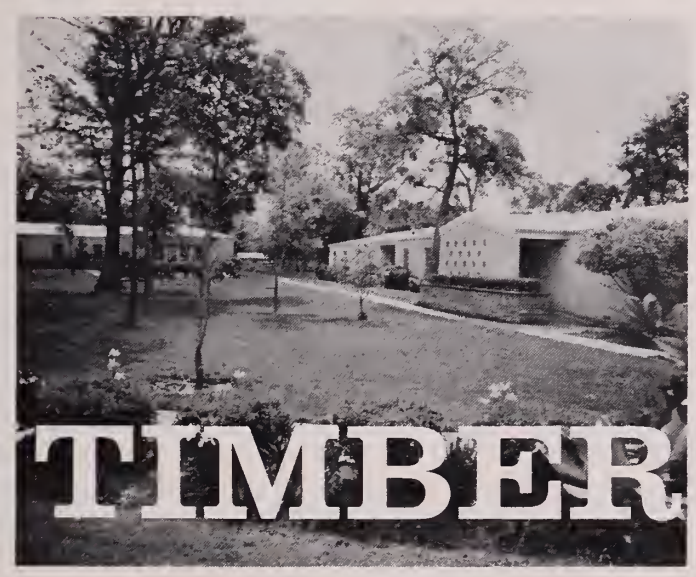
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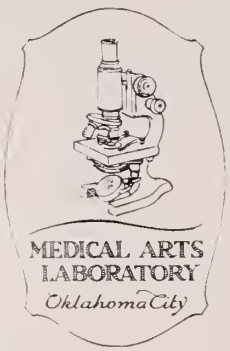
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Index to Advertisers

Ames Company, Inc. inside back
American Medical Association xviii
Arlington Hotel li
Ayerst xxxix

Beverly Hills Clinic lxi
Bone and Joint Hospital lv
George A. Breon & Co. xliv
Bristol Laboratories, Inc. xxiv and xxv
Burroughs-Wellcome & Company. Inc. xlviii

Ciba xlv
Coca Cola Bottlers lii
Coyne Campbell Sanitarium lv
Credit Service li

Endo Laboratories xxii

The First National Bank of Oklahoma City xxxviii

Geigy xxvii
General Electric xxix
Glenbrook Labs xiv
Goldfain Laboratory lvi

J. E. Hanger l

Drs. Lain, Lamb and Jones lvi
Lederle Laboratories viii and ix, 348 and 349, xxviii, xxxi, xxxiv
The Liberty National Bank of Oklahoma City xlvi
Eli Lilly & Company xvi

Medical Arts Laboratory liv
S. E. Massingill Company xiii
Melton Co., Inc. lii
Merck Sharp & Dohme, Inc. iv, xii, xxxiii
McAlester Clinic lvii
Midwest Surgical Supply Co., Inc. lx

Neurological Hospital lxi
North American Company xlix

Oklahoma Allergy Clinic lvii
Osler Radioisotope Laboratory lx

Parke Davis & Company inside front and iii
Physicians Casualty & Health Assn. lii
Physicians Laboratory lx

A. H. Robins Co., Inc. xix
Rock Haven Hospital lxi
Roerig vi, xxxii

St. Paul Companies l
Sandoz x
G. D. Searle & Company 391
Schering Corporation xv, xxx, xl and xli, liii
Seven-Up Bottlers li
Smith-Dorsey xxvi, xxxvi and xxxvii
Smith, Kline & French Laboratories outside back
Springer Clinic lviii
E. R. Squibb and Sons xi
Stout Clinical Laboratory lviii
Sugg Clinic lix

Terrell's Laboratories lx
Timberlawn Psychiatric Center liii
Tulsa Clinic lix

Wallace Laboratories insert, xliii
Wesson Oil xx and xxi
Winthrop Laboratories vii, insert, xlvii

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Order of Frequency of Presenting Symptoms in 110 Patients

Symptoms	No. of Patients	Per cent of total group
Polyuria	93	84.5
Polydipsia	89	81.0
Weight loss	47	42.7
Polyphagia	28	25.4
Anorexia	16	14.5
Lethargy	14	12.7
Enuresis	7	6.4
Vomiting	5	4.5
Irritability	3	2.7
"Craving for sweets"	3	2.7
"Sticky diaper"	3	2.7
"Strong odor to urine"	2	1.8
Glycosuria	2	1.8
Hypoglycemia	2	1.8
Personality change	1	0.9
Boils	1	0.9
Headache	1	0.9
Abdominal cramps	1	0.9

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1. Goddard, E.S.: in *Trifluoperazine, Further Clinical and Laboratory Studies*, Philadelphia, Lea & Febiger, 1959.

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BEN H. NICHOLSON, M.D.

A Tribute Page 407

FROM PARKE-DAVIS...A NEW ORAL ANTIBIOTIC

WITH EFFECTIVE ANTIBACTERIAL & ANTIAMEBIC ACTIONS

"THE FORTUNATE COMBINATION OF HIGH ANTIAMEBIC AND ANTIBACTERIAL ACTIVITY AND LOW ORAL TOXICITY MAKES PAROMOMYCIN UNIQUE AMONG THE AVAILABLE DRUGS AND SUGGESTS THAT IT SHOULD BE A USEFUL THERAPEUTIC SUBSTANCE."[1]

● **IN INFECTIOUS DIARRHEAS[2-5]** Because it is effective against gram-negative pathogens, HUMATIN has proved especially valuable in infectious diarrheas, most of which are caused by bacilli of the gram-negative group. In 221 patients with severe diarrhea, 85 per cent obtained rapid remission of symptoms with HUMATIN, and stools cleared quickly of pathogens.[2] Results in *Shigella* and *Salmonella* enteritis,[5] and in infantile diarrheas of mixed etiology[3] have been uniformly good.

● **IN INTESTINAL AMEBIASIS[2, 5-8]** HUMATIN is unusually effective in clearing all phases of intestinal amebiasis;[5] to date, more than 700 patients have been treated successfully with HUMATIN in all parts of the world. Since HUMATIN is not appreciably absorbed from the gastrointestinal tract it is not effective against extraintestinal forms of amebiasis.

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ALSO VALUABLE IN THE PREOPERATIVE SUPPRESSION OF INTESTINAL FLORA,[2] AND IN THE ADJUNCTIVE MANAGEMENT OF HEPATIC COMA [9-11]

ADMINISTRATION AND DOSAGE

(expressed in terms of base)

Condition	Dosage (daily, in divided doses)	Duration of Therapy
Infectious Diarrheas (bacillary and nonspecific)	*Adults:* 35 to 50 mg/Kg; up to 100 mg/Kg in severe cases.	Up to 7 days
	Children: 50 mg/Kg; up to 100 mg/Kg in severe cases.	Up to 7 days
Intestinal Amebiasis (acute, subacute, chronic)	*Adults:* 0.75 to 1.5 Gm.; larger doses when required.	5 days
	Children: 22 mg/Kg; larger doses when required.	5 days
Preoperative Suppression of Intestinal Flora	*Adults:* 2 Gm.	4 days
Hepatic Coma	*Adults:* up to 6 Gm., depending on degree of hepatic insufficiency and response of patient.	See literature

SIDE EFFECTS: Since HUMATIN by the oral route is virtually nonabsorbed in the gastrointestinal tract, even with exceptionally high doses, systemic toxicity has not been a problem in clinical use.[2,3,5-8] However, when doses in excess of 2 Gm. per day are given for more than three days, loose stools may develop; on doses of from 4 to 6 Gm. daily, moderately severe diarrhea has been reported in some patients. No other indications of toxicity have been observed.

SUPPLIED: HUMATIN (paromomycin, Parke-Davis) is available as the sulfate in Kapseals,® each containing 250 mg. of base, in bottles of 16. Literature supplying details of dosage and administration available on request.

REFERENCES: (1) Coffey, G. L., *et al.*: *Antibiotics & Chemother.* 9:730, 1959. (2) Personal Communications to the Department of Clinical Investigation, Parke, Davis & Company, 1959. (3) Godenne, G. D.: Paromomycin in diarrheas of infants and children, Antibiotics Annual 1959-1960, New York, Medical Encyclopedia Inc., in press. (4) McMath, W. F. T., & Hussain, K. K.: *Pub. Health* 73:328, 1959. (5) Courtney, K. O., & Thompson, P. E.: Paromomycin as a therapeutic substance for intestinal amebiasis and bacterial enteritis, Antibiotics Annual 1959-1960, New York, Medical Encyclopedia Inc., in press. (6) Shafei, A. Z.: *Antibiotic Med. & Clin. Therapy* 6:275, 1959. (7) Elias, F. L., & Oliver-Gonzales, J.: *Antibiotic Med. & Clin. Therapy* 6:584, 1959. (8) Carter, C. H.: *Antibiotic Med. & Clin. Therapy* 6:586, 1959. (9) Fast, B. B., *et al.*: *Arch. Int. Med.* 101:467, 1958. (10) Mackie, J. E., *et al.*: *New England J. Med.* 259:1151, 1958. (11) Stormont, J. M., *et al.*: *New England J. Med.* 259:1145, 1958.

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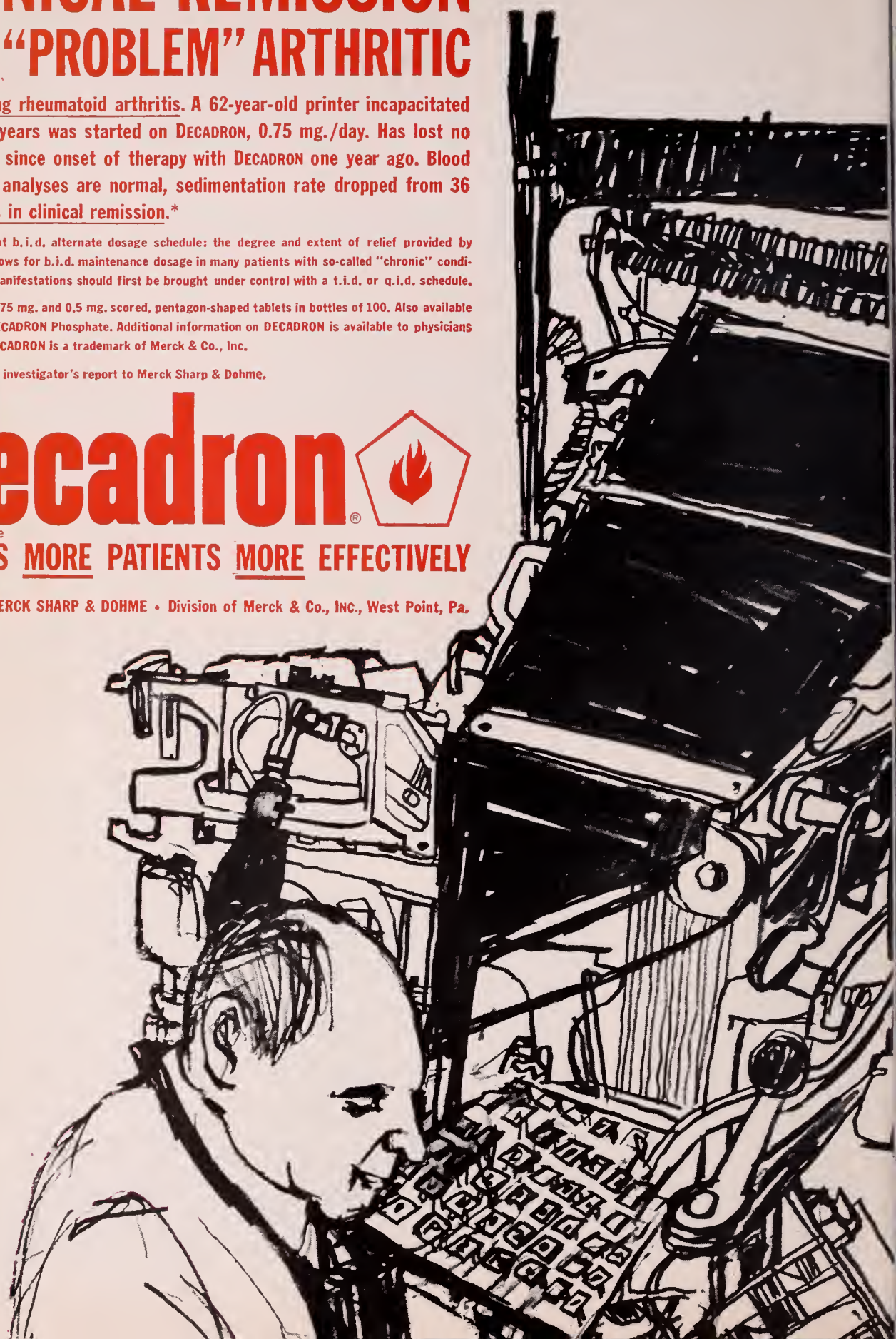
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CONTENTS

EDITORIALS

A Tribute 407
Inaugural Address 408
The Health Needs of the Aged 409
President's Letter 444

SCIENTIFIC ARTICLES

Prognosis of Renal Cell Carcinoma, *Donald W. Branham, M.D. and Willard Smith, M.D.* 412
Management of the Listless Patient, *Paul Edgar Craig, M.D.* 414
Radiotherapy of Uterine Cancer: Role of the Gynecologist, *J. M. Parrish, Jr., M.D.* 421
Fibrinolysis in Benign Prostatic Hypertrophy, *Lucien C. Kavan, M.D., Angel Bettaglio, M.D. and Hubert G. Buehler, M.D.* 426

MEDICAL CENTER

Message from the Dean 429
Tennis Elbow, *Capt. Charles R. Rockwood, Jr., USAF (MC)* 430
Abstracts 437
Faculty News 439

SPECIAL ARTICLE

Factors Relating to Physician Distribution in Oklahoma, 1950-1959, *Robert C. Lowe, M.D., James A. Hagans, M. D. and Pearl Fisher, Ph.D.* 440

NEWS

Federal Employees' Health Plan to Begin July 1, 1960 446
Council, Committees Announced for 1960-61 449
'Care for Aged' Bills Falter 453
Proceedings of the 54th Annual Session of the House of Delegates 454
Fifteen Attend Past Presidents' Breakfast 455
54th Annual Meeting Attendance, By County 456
Brill Named to SAMA Post 458
New FAA Rule Affects Physicians 458
Date Set for McAlester Symposium 459
Doctor Williams Accepts Mental Health Post 459
Bus Drivers Will Need Physical Exams 462
Have You Heard? 462
Coming Meetings 463
Auxiliary News 466
Twenty-Five Years Ago 468
Physician Placement 469
Miscellaneous Advertisements 471, Index to Advertisers lxiv

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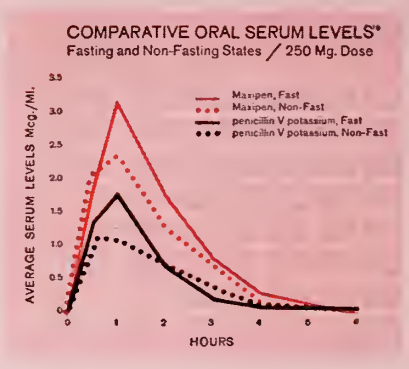
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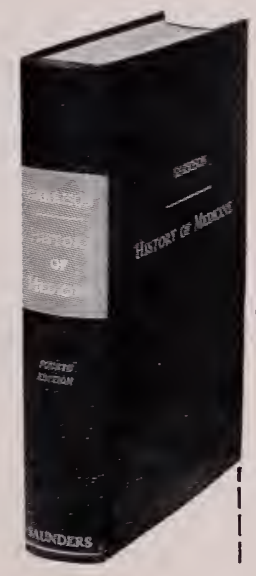
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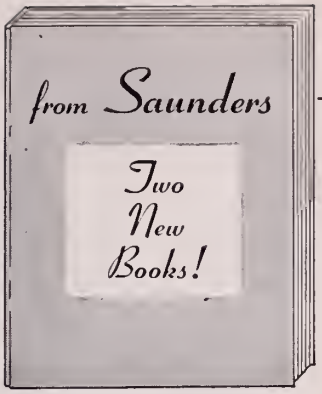
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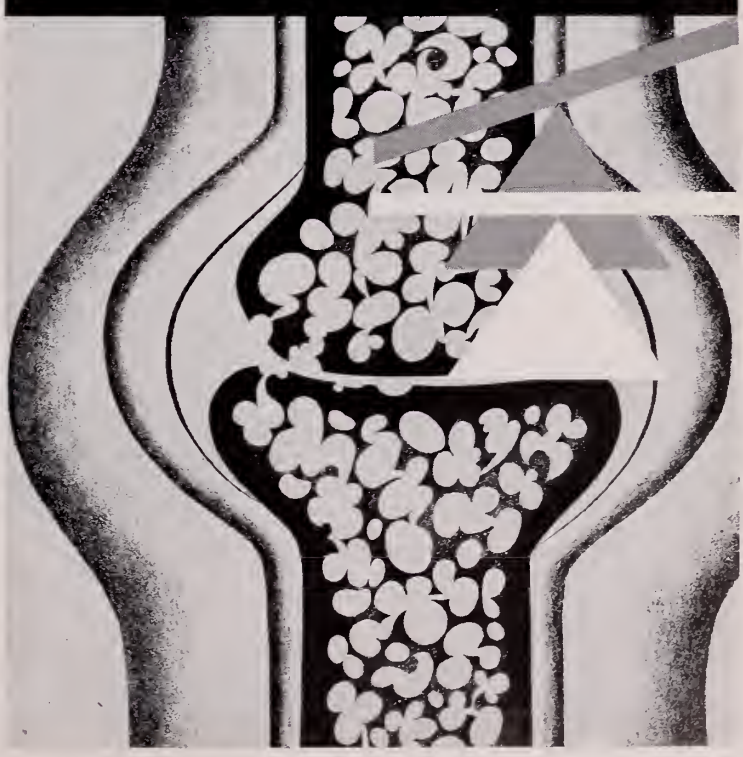
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Recent JAMA editorial statement clarifies the current controversy about dietary fats

Excerpted from the March 12, 1960, issue of The Journal of The American Medical Association:

"It is accepted generally that specific alteration in the diet will lower the concentration of cholesterol in the blood. The most effective results to date have been achieved by increasing consumption of polyunsaturated fatty acids, particularly linoleic acid. However, indefinitive and conflicting information has left much to the imagination of some food processors. Some of the largest vegetable oil processors in the United States have implied in advertisements that the cholesterol level can be lowered merely by adding polyunsaturated fatty acids to the diet. This selling campaign has created confusion among lay people, making it increasingly important that the physician clarify for his patients the conditions under which changes in the diet will be effective.

The patient should understand that if he increases his consumption of polyunsaturated fatty acids without reducing his intake of other fats, little is gained save for additional calories which could lead to obesity. A particular regimen will be effective only if polyunsaturated fatty acids are responsible for an appreciable percentage of the total fat calories. That is, they must replace rather than supplement some of the saturated fats and oils already in the diet.

* * *

Some manufacturers cite the "iodine number" of a fat or oil as evidence of the unsaturated fatty acid content of their product. This number is not a reliable indicator of therapeutic value because it measures monounsaturated and polyunsaturated fatty-acid content at the same time. A monounsaturated acid, like oleic, takes up two iodine atoms but does not affect the cholesterol concentration of the blood. A polyunsaturated acid, like linoleic, takes up four iodine atoms. In a product containing large amounts of oleic acid and small amounts of linoleic acid, the iodine number is nearly the same as it would be for a product containing little oleic acid and a modest amount of linoleic acid. Cottonseed oil has an iodine number of 110 and corn oil a number of 127; yet they each have about the same amount of linoleic acid.

* * *

Low-fat diets will not reduce the concentration of circulating cholesterol and lipoproteins as effectively as will diets containing an adequate percentage of polyunsaturated fatty acids. Weight-reduction regimens are basically low in fat, and if a lowered cholesterol level is necessary, planning must be done to maintain the proper ratio of saturated to unsaturated fats."

* * *

Herbert Pollack, M.D.
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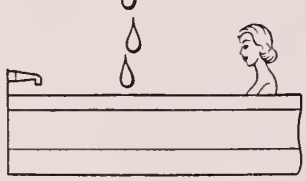
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Editorial

A TRIBUTE

THE EDITORIAL BOARD regretted to learn that Ben H. Nicholson, M.D., was relinquishing his post as Editor-in-Chief for private reasons. He first joined the Board in May 1944 and became the Editor-in-Chief in October 1954.

During the past six years, he has worked indefatigably toward his goal of making our JOURNAL second to none in its class. He spent countless hours conferring with the executive staff, with the printers and at national meetings devoted to editorial problems. He has been a Director of the State Medical Journal Advertising Bureau, Inc.

The Editorial Board, no rubber stamp individuals, cannot recall ever vetoing any proposal he has brought before it for the betterment of this magazine.

The increased size of the JOURNAL, more than doubled since 1954, its bright covers, the improved format and sharpened typography, the Medical Center section—all are due to Doctor Nicholson's efforts.

All of this work was done in addition to handling his pediatric practice, serving as First Vice-President of Blue Cross and meeting his obligations as clinical Professor of Pediatrics at the medical school.

Doctor Ben Nicholson can surely look at the current volumes of the JOURNAL of the Oklahoma State Medical Association and consider them as splendid memorials to his personal effort. The Editorial Board can only look at them as a challenge to try to meet.

The members, the Executive Staff and the Editorial Board all join in saying, "Well done, good and faithful servant."

Inaugural Address

Walter E. Brown, M.D., May 3, 1960

My respects to all the honored guests, the newly elected officers, you doctors and your wives, and all other guests. I also pay homage to Mrs. Brown and to our five children: Walter, Patricia, Phillip, Phyllis and Betty Lou and their aunt, my sister, Mrs. George Harrington of Tulsa.

This past year as president-elect has been really most enjoyable. I hate to give up the privilege of criticism that I have enjoyed. As you know, from now on the responsibilities will be mine and I will simply have to put up or shut up when the action is not favorable.

My efforts have been to serve you well thus far, although I will be the first to admit that they have sometimes been a little less than spectacular. Last October here in Oklahoma City, for instance, I attended the banquet of the Oklahoma Clinical Society as an honored guest and sat at the head table with 32 men—I was the only one not wearing a Tux. Two weeks later in Tulsa at the banquet for the Oklahoma Hospital Association, at the head table with Senator Kerr and other dignitaries I managed to be the only one who *was* wearing a Tux.

It has been interesting traveling around the State, renewing acquaintances and making new ones, and I am looking forward to getting around to more places this coming year. I have been to County Society meetings up at Vinita, over in Enid, and last week Mrs. Brown and I were guests at the Custer County Meeting out in Clinton. The job has required some travel out of State—a meeting of the A.M.A. Legislative Council in St. Louis last fall, one hurry up trip to Washington last month, etc. But it has been encouraging, wherever I have been the doctors and the great majority of the people seem to be optimistic about our profession and our nation's future.

In other State Societies an incoming president usually has the texts of remarks made by his predecessors which have been duly printed in the State Journal. We have never done this in Oklahoma, and I presume that some of you are beginning to understand why at this stage in my talk. However, it has been my privilege to know several of our past presidents personally and to admire their administrations. If I could bring to this office the equanimity of Bruce Hinson, we would be most fortunate. I wish I could offer the oratory of John McDonald . . . . . the genial and gentlemanly manner of R. Q. Goodwin . . . . . the rugged individualism of Red McClure . . . . . the fighting heart of Jack Burton . . . . . the executive skill of Clyde Mohler . . . . . the dignity of Alfred Baker. But you can't get all these qualities in one man, and I don't suppose it would solve all our problems if you could. Our picture is constantly changing, this is not news, it has been changing through the years. In this century, as Ben Nicholson points out in his thought-provoking editorial in the *Journal* this month, it started changing abruptly with the Flexner report in 1910; World War I brought new concepts to the profession; the New Deal changed our plans; the depression years; World War II; the readjustment in the postwar years, and in our most recent years, the heavy hand of organized labor and the Federal Government. We have been subjected to a subtle change through the last twenty years by the growing domain of prepaid medical care. It is obvious that the practice of medicine will continue to undergo changes, changes in type of practice, in the scientific tools and medications that we use in the profession and even in our actual working hours. It looks like the only thing we can do is keep electing guys like Baker, Gallaher or me to take time off a year at a time and try to keep a hand in the administrative aspects of medicine. We must have a worthwhile goal to strive for, even if it is altered a little bit from year to year. We must believe that our efforts are to be ultimately rewarding to mankind. Tennyson expressed this over a century ago in this quatrain from the magnificent poem, Locksley Hall:

"Yet I doubt not through the ages, one increasing purpose runs,
And the thoughts of men are widened with the process of the sons.
Knowledge comes, but wisdom lingers, and I linger on the shore,
And the individual withers, and the world is more and more."

Yes, gentlemen, in our profession of medicine, we must doubt not through the decades, one increasing purpose runs.

The Health Needs of the Aged

Recently, I asked one of my patients, a man past 70 and a person of better than average mind a question; it was about a subject of topical interest—the welfare of the aged in our population. The particular question I asked was "what in your opinion are the health needs of an older person?" This has been a most difficult question for me to answer in my own mind and it is one that can hardly be generalized about, for each health situation in the older person is an individual affair which varies from one to the other. It must be appraised and evaluated not only as it pertains to the particular organic ailment from which the person suffers, but proper medical treatment is contingent on other factors as well: the individual's environmental, economic and even psychologic background must be considered. A disability in one older person may present a critical health problem, the same ailment in another maybe of no medical consequence, demanding nothing but palliative treatment. For illustration, the individual past 70 with disability from a hip fracture that keeps him semi-bedfast may be able to get along fairly well if he has financial resources to employ nurses and aides to help him care for his basic physical needs. However, if he must live alone and depend upon himself for his physical requirements, medical care which may be of highly specialized nature will be urgently indicated just for the sake of making him physically mobile. This does not mean that one should medically neglect the older person merely because he is aged, but only that each must be individually selected for the type of treatment needed or required. Physicians often lose sight of this fact and advise treatment merely because a patient suffers from an ailment or disability. They do not take into consideration that old people should have most of the say as to whether or how they should be treated. It seems to me that the older they are the more say they should have.

But back to the question I asked—my patient hesitated in thought for a moment and finally said: "Doctor, I do not know whether I am right or not, but at the present I feel that when I consult a physician as regards any ailment or disability which after his examination proves to be of serious importance, the first consideration I request is that what he advises I should do in a medical or surgical way should not be based primarily upon whether what is done will prolong my life. I realize that I have been fortunate to enjoy a span of life this long and although I would relish a bit more living, yet, on the other hand I do not want to live merely for the sake of existence. Certainly, if simple surgical and medical procedures are available to prolong my life, this much I would expect of him. But on the other hand, if what he advises requires extensive hospitalization and radical surgical measures that may leave me physically deformed or even invalided, or what is almost as important may pauperize my family because of the expense, better even if it requires a bit of dissimulation on his part, that he tell me that he can do little in a medical way. I feel that in my particular instance of circumstances this is a falsification on his part which is morally justified.

"Doctor, I have so little time left on this earth that I feel it should not be wasted lying in the hospital just to salvage a few more problematical years of life. Extraordinary medical measures to accomplish this end seem to me unreasonable and at least a bigger price than I want to pay. Maybe I can sum up in two principles what I expect from my doctor—first I want to be free as much and as long as possible from pain; and second I want to be kept ambulatory and in a state of mental independence as long as possible. If this can be accomplished with relatively simple medical and surgical procedures, all well and good. But if it requires extraordinary means, better my physician prescribes palliative treatment and permit me to die in peace.

"May I explain what I mean by physical independence. It is not an athletic type of mobility that I seek, but only that sufficient to enable me to care for my own basic physical needs without more than token help from others; to be able to dress myself; to serve myself at the time; to tend to my toilet affairs; to be able to move from bed to chair;

and possibly walk into the open and enjoy the sounds and sights of nature around me. And then I may add I want him to maintain for me enough mental ability to give me an understanding of my environment, to know the familiar things of my life and to be able to know and communicate with the people with whom I have been intimately involved through all these years. I want to keep myself a meaningful part of this world."

He stopped and then in afterthought added, "I do not want you to make of me a vegetable, a sack of potatoes to be stored in the backroom and when decayed will be thrown out. Before that occurs, I pray you, leave me die without interference from your profession. Don't for the sake of a few more years make of my life a torment or my body a burden to be imposed upon my loved ones, those individuals whose love has made me the kind of person that I am."

I thought much of what he said, for it seemed a good answer to a difficult question, a problem which seems to resist almost all scientific study for a solution. I believe that what he said is an opinion held by most people in their advanced years of life, an opinion maybe not too well expressed but nevertheless felt. I wager that if you ask the majority of elder people the same question you will hear about the same answer. As I have said it is not a question for a sociologist to answer, it is only one which each individual can answer for himself, and that answer will depend upon the circumstances of his own life.

The health needs of the young are easier to assess from a sociologic standpoint. All that is necessary is tabulate the number of remedial disorders, ailments and disabilities which occur in a certain group of individuals of certain age and then measure the treatment accordingly. In the young two factors are important which do not prevail in the aged—the prolongation of life and the productivity of life. That is why any treatment given to the young, no matter its magnitude, is justified. The young person who suffers from a hernia or a crippled leg must be given treatment if not to prolong his life at least to rehabilitate him to a state of productivity. But in the older person these factors are not of primary importance when treatment is considered. Often old people with relatively serious health problems live much longer than most people realize, longer sometimes as their life expectancy, even without treatment.

May I cite personal experience in the medical treatment of the older individual that illustrates this principle. It has to do with the problem of prostatic cancer. Prostatic cancer is common in the elderly and always the question of radical surgery presents. We know that in cases of prostatic cancer palliative treatment may give as much as ten years of comfortable life. We know also that radical prostatic surgery will occasionally give cure from cancer but more often leaves the patient somewhat disabled. In planning any treatment for these individuals we follow the principle that when a patient's life expectancy appears to be longer than 10 years radical surgery must be considered. If, however, life expectancy is less than 10 years palliative therapy alone is advised.

Is it possible to anticipate what future facilities will be required to take care of the increasingly number of aged in our population? It is obvious that more hospital beds will be needed in the future, for as there will be more people the number of old people will increase accordingly. But in my opinion the need is far less urgent for this class of patients than most hospital administrators think; that the kind of beds most needed will be found in those hospitals which have to do with the management of the ordinary disabilities and ailments of life and which require only relatively simple medical and surgical procedures, these could be described as general practice hospitals. These hospitals will require good and adequate nursing care but certainly not specialized nursing care, nurses aides, practical nurses and such like. There will be relatively little need for personnel experienced with the heart-lung machine.

I believe at the present that most of our metropolitan areas are fairly well supplied with specialty bed hospitals enough to take

care of the present load of the sick elderly in our population. However, in certain areas of medical practice—surgery of the eye, genito-urinary diseases, dental diseases, orthopedic and neurosurgery, a few more beds may be required but the need will not be critically urgent.

I think that in anticipation of further facilities to improve the care of the aged in our communities we should endeavor to emphasize for them a non-hospitalizing type of care, a general practice type of hospital as I described or best in their own home. And it is in this latter wherein I think the visiting nurse plays a most important part. Most old people can be better cared for from a medical standpoint within the bounds of their own home if good nursing service is available. The visiting nurse system could relieve the load placed upon our specialty hospitals even at the present time if we would develop it toward its utmost possibilities.

In afterthought I think there is one exception where a deficit in the health needs for the aged exists, and it is in the field of mental health. Mental health problems produce most of the unhappy life situations in the aged; many of these individuals are emotionally mal-adjusted and if better trained personnel in this field of medical practice were available to them much more could be done for these unhappy individuals than is accomplished today.—*D.W.B.*

Letter to The Editor

Physicians Urged to Support State Question 390

July 5 is the day when Oklahoma voters will be given the opportunity to act on a constitutional amendment.

State Question 390 would give the taxpayers in each county of the state a chance to take a good long look at their public health needs and pay for the services to meet these needs—at the local county level, if they so desire. This provision for local financing is in keeping with the philosophy of government subscribed to by most physicians and others believing in the free enterprise economic system. It provides for the financing and consequent control of health services at the local level. This is in contrast to the "handout" paternalistic system of the Federal government meeting real or fancied needs as promulgated and pressured by special groups.

State Question 390, appearing in the third position on the July 5 ballot, will read:

"Shall a constitutional amendment, amending Article 10, Oklahoma Constitution, by adding thereto Section 9A, to authorize an additional county ad valorem tax levy, not exceeding two and one-half mills on the dollar of assessed valuation, when approved by a vote of the people, for a department of health, providing such department of health may be maintained with other counties, cities, towns or school districts or any combination thereof, and providing other levies and use of other funds for same purpose shall not be prohibited, be approved by the people?"

A YES vote on Question 390 does not mean that a citizen is voting himself another tax levy. It simply means that taxpayers of each county are given the privilege of studying their health problems and assessing themselves any amount up to 2½ mills—to be used for public health purposes. Without this amendment the Oklahoma Constitution does not allow the taxpayer a direct voice in the monies expended for public health purposes.

Health Department services, such as control of communicable diseases, laboratory testing, mass screening surveys, food and drug control and general sanitation inspection are available in only 48 Oklahoma counties.

In the 29 counties without a health department, none of these services are availble.

Care of the sick in the home by public health nurses, under the supervision of the patient's own physician, is not even being offered in most counties **with** a health department—because of inadequate funds.

The nine-member Oklahoma State Board of Health, five members of which are by state statute M.D.'s, has unanimously endorsed this amendment. The Muskogee County Medical Society has unanimously endorsed State Question 390.

This writer recommends that each county medical society endorse State QUESTION 390.

BERT T. BRUNDAGE, M.D.
Chairman, Council on Public Health, OSMA
Vice-President, State Board of Health

Scientific Articles

Prognosis of Renal Cell Carcinoma

A Clinical Study of 52 Patients*

DONALD W. BRANHAM, M.D.
WILLARD SMITH, M.D.

Since graduating from the University of Oklahoma School of Medicine in 1925, Donald W. Branham, M.D., has been certified by the American Board of Urology. In addition to his private practice. Doctor Branham is a Professor in the Department of Urology at the University of Oklahoma Medical Center.

Doctor Branham is a member of the American Urological Society, South Central Section, and holds a fellowship in the American Medical Writer's Association.

At the time this paper was written, Willard Smith, M.D., was taking a residency at the University of Oklahoma Medical Center. He is now a Lt. Col. with the U.S.A.F Hospital in Anchorage, Alaska.

WHAT IS the outlook for patients with cancer of the kidney following nephrectomy? Is their prognosis improved with postoperative x-ray therapy? How long do patients wait before consulting a physician for treatment after signs and symptoms of renal cancer develop?

These are some of the questions we endeavored to answer by followup examination on a selected group of 52 patients who had had nephrectomy for renal cancer four years before this study. The records were gathered from the four major hospitals within the metropolitan district of Oklahoma City. None of the patients showed recorded evidence of peripheral metastasis of their malignancy.

The types of tumors were:

Renal cell carcinoma (hypernephroma)	36
Wilms' tumor	7
Transitional cell carcinoma	6
Squamous cell carcinoma of the renal pelvis	3

Hematuria was seen most often, however, pain, weight loss and a palpable mass in the side was also seen commonly. All the children with Wilms' tumor had a palpable abdominal mass but none of the children had had hematuria. The three patients afflicted with squamous cell carcinoma of the renal pelvis had complicating calculi in the affected kidney.

Only a few of the 52 patients had been seen by a physician soon after they developed signs and symptoms which suggested the presence of cancer. A few delayed even months before seeking medical atten-

*This paper was presented at the Annual Meeting of the South Central Section of the American Urological Association, Inc., Denver, Colorado, September 28-30, 1959.

tion. Two patients procrastinated more than 18 months before obtaining medical care.

The majority of the patients, with the exception of Wilms' tumor group, were between 40 and 60 years of age; however, there were two individuals with renal cell carcinoma who were 26 and 28 years of age. The seven children with Wilms' tumor, six male and one female, were all under five years of age.

The over-all prognosis for cancer of the kidney, as shown in their series, did not seem to be favorable. Only 21 of the 52 patients were alive at the time of this study but patients with hypernephroma seemed to have a better prognosis than those with other types or renal cancer. Seventeen of the 36 patients with hypernephroma were still alive at the end of four years, a survival rate of 47 per cent. Two of the five patients with transitional cell carcinoma and two of the seven with Wilms' tumor had survived more than four years. However, all the patients with squamous cell carcinoma of the renal pelvis died soon after nephrectomy had been performed, a mortality rate of 100 per cent.

What effect had x-ray therapy on the over-all prognosis of this group of renal cancer patients? The majority did not receive radiation therapy; however, all the patients with Wilms' tumor were treated with radiation. Of the 16 patients with renal cell carcinoma who had received x-ray therapy after operation only five had survived more than four years. However, of the 32 untreated patients 14 were still alive at the time of this investigation.

Comment

This report compares somewhat with previous and similar clinical studies on renal cell carcinoma, particularly in showing that although cancer of the kidney has a relatively unfavorable outlook so far as eventual cure is concerned a sizable number of these patients manage to live symptom free for a relatively long period following nephrectomy.

Undoubtedly, the unfavorable prognosis observed in renal malignancy is due partly to the fact that its signs are not recognized soon enough. The survival rate in this series probably could have been improved if more of the patients had sought medical attention earlier. For example, the significance of urinary bleeding should be re-emphasized to both patient and doctor. Because it is the most frequent sign for cancer of the kidney, the presence of blood in the urine should never be dismissed as of no significance.

This study also suggested that there may be some relation between squamous cell carcinoma of the renal pelvis and stone disease; long standing renal calculi may in a few instances aggravate or even initiate the neoplastic process.

The prognosis of renal cancer seems directly related to its type. The best outlook appears to be in patients with hypernephroma; the poorest in patients with squamous cell carcinoma of the renal pelvis. The prognosis for Wilms' tumor and transitional cell carcinoma seems to be of less favorable degree than hypernephroma.

How much value is there in x-ray therapy? It is generally agreed that x-ray therapy is useful in Wilms' tumor because of its embryonic character. However, in our opinion it is questionable that radiation therapy contributes much to the treatment of other types of renal carcinoma.

1200 N. Walker, Oklahoma City, Oklahoma

Management of The Listless Patient *

PAUL E. CRAIG, M.D.

Paul Edgar Craig, M.D., graduated from Northwestern University School of Medicine in 1930. He is presently in general practice in Tulsa, Oklahoma.

Doctor Craig is a member of the American Board of Abdominal Surgery, the American Academy of General Practice, the American Anthropological Association, a Fellow of the American Geriatrics Society and a Fellow of the American Medical Writer's Association.

Doctor Craig is the author of a recent book, "Know Your Doctor."

LISTLESS PATIENTS are those individuals who complain of symptoms of muscular weakness due to loss of muscular tone, poor to complete loss of appetite, lassitude, day time drowsiness, slow speech, fatigue, irritability, and loss of weight. Examination of these patients presents a conspicuous absence of specific physical and laboratory findings. A complete list of symptoms referrable to the functional disease entity known as the "listless patient syndrome" is contained is shown in Table I.

*Presented at the Twelfth Annual Scientific Assembly of the Illinois Academy of General Practice in Chicago, Illinois on November 10, 1959.

SYMPTOMS

1. Muscular weakness due to loss of muscular tone.
2. Easy fatigability.
3. Patient arises in the morning as tired as when he retired at night.
4. Poor to complete loss of appetite.
5. Restless sleeping accompanied by nightmarish dreams or absolute insomnia.
5. Daytime drowsiness.
7. Lack of nervous energy associated with nervous hyper-irritability.
8. Loss of weight (16-18 lbs.)
9. Indigestion accompanied by vague abdominal discomfort.
10. Constipation.
11. Hat band (vertex headaches).
12. Dizziness with sudden change of position.
13. Inability to concentrate and to remember recent events clearly.
14. Aching muscles especially those of the arms, shoulders and legs.

TABLE I

Upon hearing some or all of these complaints, the physician immediately concludes that here is a chronically ill person or a sympathy-seeking neurotic. A very concerned and deliberate attitude should be adopted in the history taking and examination of these patients. A complete list of the physical findings common to the "listless patient syndrome" is set out in Table II.

PHYSICAL FINDINGS

1. Sallow, pasty skin.
2. Sunken eyes.
3. Hypotension with narrowing of the pulse pressure due to low systolic tension.
4. Slow, dull heart beat. The pulse is easily compressible and unstable.
5. Exaggerated patellar reflexes.
6. General abdominal tenderness, especially of the left lower quadrant.

TABLE II

The most frequent observations are sunken eyes, a pasty skin, hypotension, and a slow, dull heart beat. The listless patient usually presents a normal blood picture. Occasionally there is a slight hypochromic anemia (10-11 grams of hemoglobin) and a clinically significant serum hypoproteinemia of 4.8 to 5 grams. However, the albumin and globulin ratio remains unchanged. The blood glucose, N.P.N., and calcium determinations are within normal limits and liver function tests are normal.

From the past case histories of these patients and results of complete physical examinations I have categorized them into the following seven groups as listed in Table III.

CATEGORIES OF THE LISTLESS PATIENT	Cases
1. Hidden Hunger (hypovitaminosis)	22
2. Protein Depletion	18
3. Aching back, muscular weakness and easy fatigability (osteoporosis)	20
4. Generalized bone and joint pains—anorexia. (Osteolytic metastatic breast cancer)	2
5. Weight Retardation	16
6. Prematurity	2
7. Senility	4
60 Males—24 Females TOTAL	84

TABLE III

Some or all types of these listless patients are daily visitors to the doctor's office. In fact, like the poor, they are with us always. Time and again, they consult us relating their complaints and no doubt each of us at some time has been haunted to the point of frustration by the classical medical nagger. There is, however, a tangible and underlying tissue deficiency common to all patients in this group. Their thinness, to the point of emaciation, is indicative of this fact, despite a history of an adequate diet and vitamin intake. The possibility that many of these patients lack the ability to utilize protein and certain nutritional supplements may account for this condition. The more one studies this group of patients, with a paucity of objective physical findings, the more one is led to believe that there is a definite need of a biologic stimulant for increasing weight and energy.

The idea of hormonal stimulation is not new. In 1889 Brown-Sequard,[1] a noted physiologist, administered testicular extracts to himself and felt that he acquired an increase in vigor and a greater capacity for work. Since then, chemists have striven to isolate, purify, and synthetically prepare tissue-building substances without androgenic activity.

At present, our listless patients are being treated with a tissue-building substance: Nandrolone Phenpropionate.* It is a new steroid in that a methyl group at position 19 is absent and is esterified with the phenylporpionate radical at carbon 17. This results in an increase of the anabolic activity and a lack of the characteristic androgenic function. The chemical name for Nandrolone Phrenpropionate is 19-nor-4-androstene-17$_B$-ol-3-one-B-phenlypropionate. Its structural formula and comparison with other androgens is listed in Table IV.

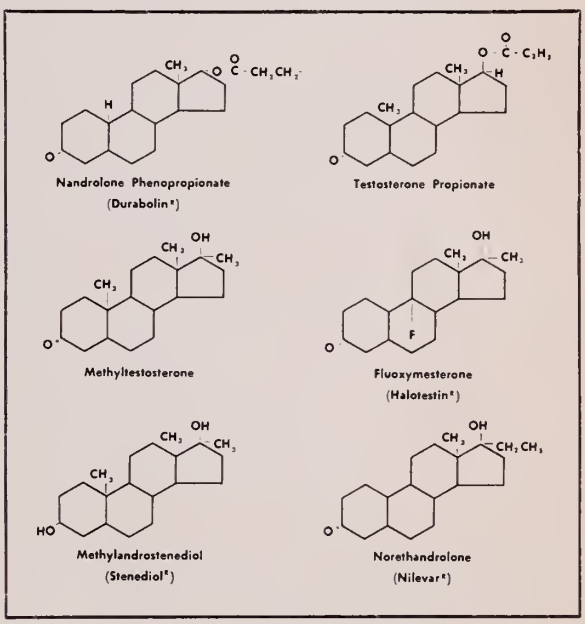


TABLE IV

Comparison of tissue building substances with respect to their route of administration, dosage, and effectiveness is shown in Table V.

Banghart[3] reported an increase in strength, mobility of joints, and complete relief of pain in patients suffering from osteoporosis who were treated with Nan-

*DURABOLIN, Organon Inc.

COMPARISON OF TISSUE BUILDING STEROIDS[2]

Androgen	Route	Dose	Relative Effectiveness Androgenic	Relative Effectiveness Anabolic	Duration of Action	Vehicle
Testosterone Propionate	I.M.	50 mg.	100	100	3-4 days	Oil
Testosterone Propionate	Buccal	50 mg.	50	50	1 day	Propylene Glycol
Crys. Testosterone	Buccal	50 mg.	50	50	1 day	Compressed Tablet
Fluoxymesterone	Oral	10 mg.	100	100	1 day	Compressed Tablet
Methyltestosterone	Buccal	50 mg.	100	100	1 day	Propylene Glycol
Methyltestosterone	Oral	50 mg.	50	50	1 day	Compressed Tablet
Norethandrolone	Oral	50 mg.	30	100	1 day	Compressed Tablet
Nandrolone Phenpropionate	I.M.	25 mg.	5	200	7-10 days	Oil

TABLE V

drolone Phenpropionate. No evidence of virilism or fluid retention was noted in therapeutic doses.

Payne[4] in his lecture at the Kentucky Academy of General Practice reported an increase in appetite, weight, and strength in his series of patients treated with Nandrolone Phenpropionate. No appreciable evidence of toxicity or virilism was observed.

Reports from other investigators* in the European literature have shown that Nandrolone Phenpropionate produced weight gain through a protein sparing action which at times is in conjunction with calcium retention. The composite of their reports, from approximately 750 patients treated with Nandrolone Phenpropionate, indicates that it is a potent tissue-building substance with minimal undesirable reactions.

These reports confirm my findings with the use of Nandrolone Phenpropionate in the treatment of the listless patient.

Prior to instituting therapy, Nandrolone Phenpropionate was clinically tested for toxicity and androgenic effects. The following tests were employed:

1. Effect of Lactation: Four mothers, postpartum four hours, were given a single injection of 200 milligrams of Nandrolone Phenpropionate. No suppression of lactation occurred immediately or delayed. Four other mothers were given a single 200 milligram dose of Testosterone Propionate at the same time postpartum, and in these suppression of lactation was complete and satisfactory.

2. Effect in Advanced Cirrhosis with Ascites: A white male 62 years of age having what was believed to be a terminal case of Laennec's Cirrhosis, associated with portal hypertension and a massive ascites, had a diagnostic confirmation by needle biopsy. This patient was given injections of 100 milligrams of Nandrolone Phenpropionate every fourth day for a total of four doses, and then 50 milligrams twice a a week for ten weeks. Liver function tests at beginning of treatment showed definite intrahepatic obstruction. Liver function tests after treatment showed a 30 to 40 per cent return of normal function to the liver. The patient had required paracentesis once a month from October to January when this therapy was started. The next paracentesis was done in March 1959 (19 liters withdrawn) and no paracentesis has been required since.

   Case of Biliary Cirrhosis: A 28 year old white female having biliary cirrhosis and an icteric index of sixteen, was given 50 milligrams of Nandrolone Phenpropionate every ten days. While she was being so treated, the jaundice, clay colored stools, and bile in urine disappeared. The diagnosis of biliary cirrhosis was made by direct liver biopsy when the common duct was explored for a possible calculus.

3. Effect of Mammary Carcinoma: A 62 year old white female suffering from

*See Bibliography 5, 6, 7, 8, 9, 11, 12, 13, 20, 21.

mammary carcinoma with abundant axillary metastasis had a left simple mastectomy. This was followed by a series of thirty-five consecutive cobalt radiation treatments to all quadrants of the left pectoral girdle. She was given 50 milligrams of Nandrolone Phenpropionate three times a week for two weeks and then 50 milligrams every ten days with complete relief from leg and arm pains.

From these brief studies, with larger doses, I concluded that Nandrolone Phenpropionate is not likely to suppress lactation, as did Testosterone Propionate, and that liver function improved even in the presence of liver pathology. Also, remarkable relief from pain was obtained in patients suffering from mammary carcinoma, which confirms the work of Von Dommelen,[13] Gerbrandy,[14,15] Den Ottolander,[16,17] and Labhart,[18] who reported complete relief from pain, a pronounced protein sparing effect, and a reduction of excessive urinary excretion of calcium that often occurs in such patients. Table VI shows the comparison of Nandrolone Phenpropionate with Testosterone Propionate on calcium excretion in Gerbrandy's patients.

With the safety of Nandrolone Phenpropionate having thus been established, it was administered to 84 individuals of both sexes, each of whom had 50 per cent or more of the symptom-complex referred to as the listless patient syndrome. Approximately 650 injections were given over a 12 month period in the categories as described.

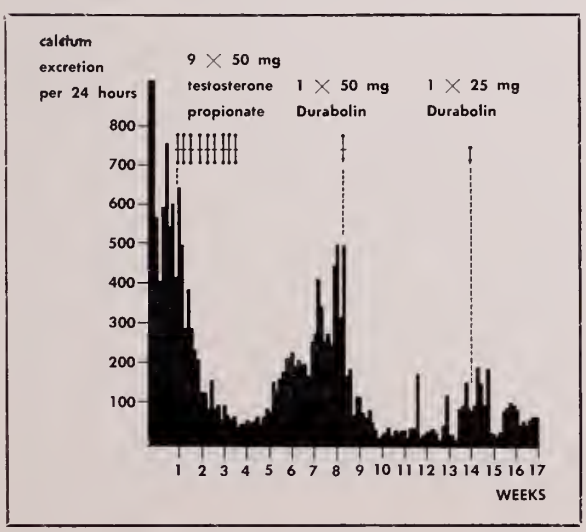


TABLE VI

HIDDEN HUNGER (Hypovitaminosis)—22 patients

This group, comprised principally of adolescent food faddists who complained of weakness and loss of physical stamina and who showed loss of weight due to malnutrition and hypovitaminosis, was treated with Nandrolone Phenpropionate. The age range was 13-19 years. These young people subsisted chiefly on candy, hot dogs, potato chips, and soda pop between meals and ate only trifling amounts of their regular meals. We prescribed an initial dose of 25 milligrams of Nandrolone Phenpropionate every 5 days for 4 doses. Then once a week until their weight gain reached the median percentile for height and age. The average weight gain for these patients was 6 pounds every 3 weeks. One girl age 17, 5′6″; weighing 98 pounds received a total of 175 milligrams of Nandrolone proprinate during 4 weeks of treatment. Her weight gain was 16 pounds within 35 days. Simultaneously with injections of Nandrolone Phenpropionate we prescribed a daily oral intake of protein 100 grams, carbohydrate 200 grams, and fat 130 grams or a total of 2,000 to 2,200 calories per day. A therapeutic vitamin capsule was also prescribed and taken each morning.

PROTEIN DEPLETION—18 Patients

For those individuals with proven serum hypoproteinemia of less than 5 grams of serum propetin per 100 cc. of blood, we prescribed a daily nutritional supplement of protein 100 grams, carbohydrate 235 grams, and fat 130 grams, or a total of 2,450 calories in conjunction with an initial dose of 50 milligrams of Nandrolone Phenpropionate. Five days later 25 milligrams of Nandrolone Phenpropionate was prescribed and then 25 milligrams every 5-7 days until the serum protein level approached 7 grams per 100 cc. of blood. The average time to restore serum protein levels to normal was about 4 weeks. Improvement was apparent one week after the first injection. This was accomplished by a weight gain of from 6 to

15 pounds in four weeks which was retained after discontinuance of Nandrolone Phenpropionate.

OSTEOPOROSIS—20 Patients

Causes of osteoporosis were:

	Cases
1. Postsurgical menopausal states	8
2. Prolonged treatment with ACTH and Cortisone for arthritis	4
3. Cushing's Syndrome	1
4. Hypovitaminosis	3
5. Malnutrition	4

Each of these patients was given an initial intramuscular injection of 100 milligrams of Nandrolone Phenpropionate followed by 50 milligram doses every seven days for three weeks and then weekly injections of 25 milligrams for one month. There was a marked improvement in all patients having painful joints especially of the lower lumbar and sacral areas. The average urinary calcium of the 20 patients, decreased from 1.5 grams in 24 hours to a normal of 0.1 grams. The inorganic serum calcium was decreased from 12.8 to 9 milligrams within the first two weeks of treatment.

OSTEOLYTIC METASTATIC BREAST CANCER—2 Patients—Ages 48-52

One patient had had a radical mastectomy and had had a surgical menopause (15 years ago following a sub-total hysterectomy) and the other with a simple mastectomy and axillary metastasis had a radiation castration.

Each was given an initial dose of 100 milligrams of Nandrolone Phenpropionate every five days for four doses. Each gained weight, had improvement of mood, a general feeling of well-being and was relieved of pain in all long bones of the body.

WEIGHT RETARDATION—16 Patients

This occurred in children of both sexes ranging from 3 to 5 years of age. Initial doses of 25 milligrams of Nandrolone Phenpropionate then 12.5 milligrams were administered at seven day intervals for a total of six injections. The average gain in weight was 4½ pounds during the six week period. Three of these patients had clinical rickets substantiated by x-ray, serum alkaline phosphatase levels and the elevation of and lowering of the serum phosphorus. These patients, in addition to Nandrolone Phenpropionate injections, were given 10,000 units of Vitamin D and 25,000 units of Vitamin A orally three times a day. Improvement of all patients was manifested by:

1. Increase in weight.
2. Interest in their surroundings.
3. Disappearance of the bluish color of the sclerae.
4. Alertness.
5. Desire to play.
6. Marked increase in appetite.
7. Marked reduction of restlessness and improved sleeping habits.
8. Disappearance of head sweating.
9. Pronounced increase in body vigor and vitality.

PREMATURITY—Two Infants

One infant born in the 30th week of pregnancy weighed 2 pounds 8 ounces, and another born in the 35th week of pregnancy weighed 3 pounds 2 ounces. After the first week of birth each was given 8 milligrams of Nandrolone Phenpropionate and thereafter every five days for a total of six injections. At the end of two weeks these infants had gained an average of one ounce a day during the time they were under treatment. They increased in vigor and vitality and the sucking reflexes were markedly stimulated.

SENILITY—4 Patients—3 Males—1 Female

These patients were given oral pancreatic enzymes, dilute hydrochloric acid, vitamin B_{12}, and diets high in protein and caloric content. They were given 25 milligrams of Nandrolone Phenpropionate parenterally twice a week for three weeks and then 25 milligrams every ten days for a total of ten injections. At the end of treatment each showed an increase in strength, muscle tone, and sense of well-being including a return of appetite. The average weight gain was 4 pounds in six weeks.

POSOLOGY—(Dosage schedule)

Infants: From first week of birth to first year were given 8 milligrams of Nan-

drolone Phenpropionate weekly, times six doses or a total of 48 milligrams.

Children: Three to five years of age, manifesting a definite weight lag, lack of energy and growth, poor appetite, and diminution of physical vitality, were given 12.5 milligrams every five days for four doses, then 12.5 milligrams every 7-10 days for a total of six doses or a total of 125 milligrams.

Adolescents: Thirteen to nineteen years of age, manifesting loss of weight, loss of appetite, hypoproteinemia and lack of the normal daily essential amino acid intake, were given 25 milligrams twice a week for four doses and subsequently 25 milligrams once a week for four weeks or a total of 200 milligrams.

Adults: Dosage was adjusted according to the following conditions:

Osteoporosis—Initial dose of 100 milligrams followed by weekly injections of 50 milligrams times three, then 25 milligrams a week times four or a total of 350 milligrams.

Osteolytic Metastatic Breast Cancer—Initial dose of 100 milligrams every five days times four (400 milligrams) then 50 milligrams weekly until leg and arm pains disappeared which was an average of six weeks (300 milligrams) for a total of 700 milligrams.

In this dosage, no edema of the face or ankles was noted and there was only slight deepening of the voice and appearance of hair on the upper lip. These side effects disappeared after treatment had been discontinued for six weeks.

Senility—Twenty-five milligrams twice a week for three weeks or six injections then 25 milligrams every ten days for a total of ten injections or a total of 400 milligrams in nine weeks.

During the course of this investigation I attempted to list all the possible side effects that could potentially occur from an accumulated dosage of 200 milligrams or more of Nandrolone Phenpropionate; however, only eight patients (10 per cent), (6 children and 2 adults) experienced any side reactions.

Table VII shows the composite of these reactions.

SIDE EFFECTS WITH ACCUMULATED DOSAGE OF 200 MILLIGRAMS OR MORE OF NANDROLONE PHENPROPIONATE

1. Generalized hirsutism, thinning of the scalp hair and increase of facial hair (metastatic breast cancer)	2
2. Oily skin and acne	0
3. Enlargement of the clitoris (femals under 3) (reduced when drug withdrawn)	2
4. Cessation of menses	0
5. Increased libido	0
6. Premature closure of the epiphyseal line	0
7. Gynecomastia	0
8. Decreased spermatogenesis	0
9. Priapism (boys under 5)	2
10. Voice change	2
11. Hypercalcemia	0
12. Hyperlipemia	0
13. Jaundice	0
14. Oliguria	0
15. Cardiac decompensation	0

Discussion

In this series of 84 patients, 8 or approximately 10 per cent had transitory androgenic reactions consisting of huskiness of the voice, hair growth on the upper lip and chin, priapism and hypertrophy of the clitoris. Adult males manifested no untoward effects whatsoever. Two boys ages 3 and 5 developed a priapism which was transitory and disappeared after the drug was discontinued.

The tissue-building effects of Nandrolone Phenpropionate were evident by weight gain in all patients, which was maintained after discontinuance of therapy.

There was a beneficial effect on the psyche and on the entire muscular and vegetative nervous systems, brought about by an increase of body weight, enhanced vitality, stamina, and a marked show of appetite. Liver function tests remained normal even after large doses of Nandrolone Phenpropionate. During the administration there was an increase of serum protein to normal values.

Following eight to ten weeks of therapy, the patient tends to retain a positive nitrogen balance with no appreciable effect on sodium excretion or retention.

This study is being continued on surgical patients, especially those who have hypoproteinemia, are malnourished and are therefore poor operative risks. Nandrolone Phenpropionate, 50 milligrams, is prescribed one week prior to operation and every third day following operation for the purpose of promoting rapidity of wound healing and strengthening the incisional scar that forms on the sixth or seventh day.

Summary and Conclusions

Eighty-four patients, classified as "listless," suffering from osteoporosis, protein starvation, metastatic breast cancer, weight lag, hidden hunger, prematurity, and senility were treated with Nandrolone Phenpropionate.

The results, after twelve months of therapy, indicate:

1. The average weight gain from treatment with Nandrolone Phenpropionate is: for adults 8 pounds within 4 weeks; children (3 to 5 years), 4½ pounds in 6 weeks; adolescents (13-19 years), 6 pounds every 3 weeks; infants (premature), 1 ounce daily after 2 to 4 weeks of therapy.
2. Serum protein levels increased 2 mg. within 4 weeks.
3. Relief of pain, experienced by patients with metastatic breast cancer, occurred within 1 week of therapy.
4. Senile convalescent patients had an increase in strength, muscle tone, appetite, and sense of well-being.
5. Toxicity and side effects test proved that: (a) Nandrolone Phenpropionate did not cause suppression of lactation from a dosage eight times the maintenance dose. This was indicative of its lack of androgenic activity. (b) Liver function improved even in the presence of liver pathology (Laennec's Cirrhosis).
6. In this series there were no local reactions or systemic allergic manifestations to repeated injections of Nandrolone Phenpropionate.

It is my conclusion that Nandrolone Phenpropionate is an excellent preparation for tissue building due to nitrogen retention and has minimal transitory side effects and possesses a wide range of safety.

Addendum

Since completion of this paper twenty major surgical cases have been treated pre and post-operatively with Nandrolone Phenpropionate. All patients (18 females and 2 males) showed a marked improvement of psyche, had a general increase of well-being, felt stronger, ate better and were dismissed two days earlier from the hospital than those in the control group.

Cholecystectomy	4
Total Abdominal Hysterectomy	8
Appendectomy	6
Gastric Resection	2

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2732 E. 15th, Tulsa, Oklahoma

Radiotherapy of Uterine Cancer: Role of The Gynecologist

J. M. PARRISH, JR., M.D.

J. M. Parrish, Jr., M.D., graduated from the University of Tennessee College of Medicine in 1934 and has since been certified by the American Board of Obstetrics and Gynecology. In addition to his private practice, he is Assistant Professor of Gynecology at the University of Oklahoma School of Medicine.

Doctor Parrish is a member of the American College of Obstetricians and Gynecologists, the Central Association of Obstetricians and Gynecologists and a Fellow in the American College of Surgeons.

PROGRESS in the radiotherapy of malignant neoplasms of the uterus in the past decade rivals the advances made in other phases of medicine. The techniques of radiation therapy today are as superior to those of eighteen to twenty years ago as is today's total to yesteryear's subtotal hysterectomy. Using the older irradiation techniques today would be equivalent to adhering to outmoded subtotal uterine resections. An enormous literature on this subject has accumulated in the past ten years. Older writings on the treatment of cervical and endometrial cancer are now of only historic interest.

Much of the advance in this therapy must rightfully be credited to the radiologist, the radiotherapist and the physicist; but the gynecologist must avail himself of these advances in rendering optimal treatment to his patient with cervical and endometrial carcinoma. Adequate treatment of these patients requires, in addition to all the essential radiologic paraphernalia, close cooperation among gynecologist, radiologist, pathologist and physicist. Oklahoma is fortunate in having adequate modern equipment and competent professional personnel; and it is the duty of all physicians to utilize these facilities in giving their patients treatment as effective as that available anywhere in the country.

Small and isolated groups are treating carcinoma of the cervix surgically; but the accepted methods of choice are radiotherapy for cervical cancer and radiotherapy combined with surgery for endometrial cancer.

This discussion of these methods of therapy is based on personal experience and on reports by leading authorities in this field; radiologists, gynecologists, and pathologists —Kottmeier, Schmidt, Arneson, Nolan, Fletcher, Galvin, Moss, Henriksen, Morton, Meschan, Ackerman and others.

Based on thousands of cases, the five-year salvage rate in Stage I cervical carcinoma is 81 per cent and in Stage II 67 per cent. The eleventh Annual Report of Results of Treatment of Carcinoma of the Cervix gave 70 per cent as the five-year salvage rate in 45,000 Stage I cases treated in 86 different institutions. Kottmeier recently reported 88.2 per cent in Stage I and 67.8 per cent in State II among 1,464 cases.

Presented before the Oklahoma City OB and GYN Society. February 17, 1960.

The major factors to be considered in planning radiotherapy for cervical cancer are: (1) the site of origin and the route of spread, (2) the radiation tolerance of neighboring organs, and (3) the radiosensitivity of the primary lesion and its extensions. These factors vary from patient to patient. There are also variations in the anatomy. This necessitates the availability of a wide assortment of equipment for proper treatment of the individual patient. It is unfair to make the patient fit the applicator rather than the applicator to fit the patient just because the proper applicator is not available.

As early as 1938, Arneson favored the interstitial radiation therapy of cervical carcinoma, and in 1952 reported the use of an expanding, fixed tandemovoid colpostat. Other fixed ovoids were devised by Ernst, Fletcher, and Nolan. It is almost universally agreed today that in treating cervical cancer the single, loose, unattached ovoids of the Manchester type are obsolete. Meschan, in a symposium on supervoltage therapy at the Oakridge Institute of Nuclear Studies, stated that if a patient measures no more than 20 centimeters in the anterior posterior diameter she may be treated adequately with the 250 kilovolt machine, but that in all others radium therapy should be supplemented with cobalt-60 or supervoltage x-ray machines.

Most authorities today prefer a tandem in the cervical and uterine canal supplemented by interstitial needle irradiation, when fixed applicators cannot be used in radium therapy. It is generally agreed that the uterine tandem alone, properly loaded in Stages I and II will irradiate the primary lesion adequately and at point A; it will deliver approximately 20 per cent gamma roentgens to point B. Following this, additional dosage is supplied by cobalt-60 or supervoltage irradiation. It is also accepted practice, based on pathologic studies of reported biopsies, to give the radium in divided doses, from seven days to ten days apart. In large cauliflower lesions and those of Stage I and II associated with parametrial infection, it is advisable to give at least a part of the external irradiation prior to the use of radium. But in all other lesions of Stage I and II radium is used first. Lesions in Stages III and IV are best treated first with external irradiation and then with radium according to their response.

An error of possibly 25 per cent is universally admitted in the staging of cervical carcinoma according to the following internationally accepted classification:

Stage O Carcinoma in situ, a preinvasive lesion.

Stage I Carcinoma strictly confined to the cervix.

Stage II Carcinoma which has extended beyond the cervix but has not reached the pelvic wall or involved the lower third of the vagina.

Stage III Carcinoma which has reached the pelvic wall of the lower third of the vagina.

Stage IV Carcinoma which involves the bladder or the rectum or which has extended beyond the link of Stage III.

Doseage of radiation cannot properly be expressed in milligram hours any more than can the efficacy of hysterectomy be expressed in terms of time spent on the operation. The dosage is properly expressed in gamma roentgens delivered to point C, A, B and at times to point W, the time required to deliver these dosages, whether they are fractionated, etc. Gamma roentgens from radium, from cobalt-60 and from supervoltage deep therapy x-ray machines are synonymous; but not from the 250 volt machine—an additional reason for preference of cobalt-60 and supervoltage for this purpose.

Cancerocidal dosages expressed thus in gamma roentgen (or simply roentgens) are as definite and mathematical as dosing medicines in minims, cubic centimeters, grams and ounces; expressing irradiation dosages in milligram hours is equivalent to telling the patient to take "a dose" of medicine.

The uterine cancer tolerates higher dosages of radium irradiation than any other

comparable tissues of the body—as high as 20,000 to 30,000 gamma roentgens at the radium source, point C (0.7 cm. lateral to the center of the cervical canal). Cancerocidal doses lie between 5,000 and 14,000 gamma roentgens—a wide margin of safety to the normal cervical tissue. A reasonable initial dose is 6,500 to 8,000 roentgens, according to the applicator used, to point A (2 cm. lateral to the center of the cervical canal at the external os or, more correctly, 2 cm. above the colpostat in the vaginal fornix).

The optimal dose at the rectal wall is 4,500 gamma roentgens and the tolerance dose is but little greater; so much larger doses should not be used if rectal injury is to be avoided. Similarly, the optimal dose at the posterior bladder wall, about 5,500 gamma roentgens, is but little less than the tolerance dose. Knowledge of these facts is essential to proper vaginal tracking for distance screening of these structures. Even with careful packing the bladder may be only a centimeter from the tandem or only a millimeter from a needle at twelve o'clock in the periphery of the cervix.

Unlike x-ray tubes it is unnecessary to calibrate radium sources repeatedly, because a definite quantity of radium under a definite set of conditions always delivers the same amount of radiation. The half-life of radium is approximately 1700 years, so that once the output of a radium source has been determined, it will be known to remain the same for many years. Dosages delivered by a variety of applicators may be calculated and tabulated, once and for all, from data obtained by physician measurements. These facts introduce the most important part of this presentation.

For proper management of cervical carcinoma, a knowledge of the applicator used is essential—its loading, the filtration used, the active length of the source, the gamma roentgens delivered to the lesion (point C, for practical purposes and to points A and B, and in more cases to point W).

Point A is 2 cm. lateral to the center of the external os and 2 cm. superior to the ovoid in the lateral fornix. Point B is 5 cm. lateral to the center of the cervical canal at the external os. Point C is 0.7 cm. lateral to the center of the external os. Point W is 6 cm. lateral to the center of the cervical canal at the external os and represents the lateral pelvic wall and the region of the obturator nodes.

Upon completion of treatment with radium 9,000 or more gamma roentgens will have been delivered to the lesion; 6,000 to 8,000 to point A; and 1,500 to 2,000 to point B. The radiologist is then in a position to deliver an additional 3,500 to 4,000 roentgens to point B and W and to the tissues between these points and point A, so that finally the patient will have been adequately irradiated with cancerocidal dosage. The radiologist must be told the dosage previously given so that he may properly screen the midline for width of 4 or 5 cm. to minimize further radiation exposure to the bladder and rectum.

Although the basic technical procedures of measurements, etc., have been worked out by the physicist and the radiologist, the gynecologist should be familiar with what has been done and with the various factors involved in radiotherapy of the carcinomatous cervix. The physicist informs us that a tubular source of radium 2 cm. in active length, filtered through 0.5 mm. of platinum will deliver 6.25 gamma roentgens at a depth of 1 cm. per milligram hour, 1.96 gamma roentgens at a depth of 2 cm. per milligram hour, and 0.33 gamma roentgens at a depth of 5 cm. (at point B) per milligram hour. Hence, 5,000 milligram hours yield 1,650 gamma roentgens to point B—the lateral pelvic wall. To obtain a cancerocidal dose, therefore, the radiologist must deliver an additional 3,500 roentgens to this area. This calculation applies to a simple source placed in the cervical canal and the uterine cavity. This dose at point B and to tissues between point B and A is supplemented by a radium source in the polpostat using a fixed applicator placed in the vaginal fornix.

When the anatomic situation precludes the use of a fixed applicator with fixed ovoids, the tandem source is supplemented with interstitial needles in the cervical periphery—lateral rather than mesial. The tan-

dem alone, when properly loaded is adequate in Stages I and II. The colpostat and needles are used to compensate for the deficiency at points A, B, and W, in intervening tissue without increasing the midline dose to the bladder and rectum beyond tolerance.

Lymph nodes in the region of point A, B, and W must be considered. These are involved in 20 to 25 per cent of cases of Stages I and II. Taussig, Morton, and Henriksen report finding cancer in nodes of 30 to 35 per cent of non-irradiated and in 10 to 14 per cent of irradiated patients. These reports were made before the availability of supervoltage x-ray machines and cobalt-60 and largely before our present knowledge of gamma roentgen dosage. It will be interesting to note what these new modifications will accomplish.

In the management of endometrial (or corpus) uterine cancer, Schmidt reports a five-year survival rate of 81 per cent and adds that there is a 20 per cent improved rate in patients given adequate preoperative irradiation. The corresponding figures of Kottmeier are 84 and 15 per cent. He states radiotherapy and surgery to be essential in carcinoma of the endometrium. The opinions of these two authorities are concurred in by most workers in this field—cancerocidal doses of radiation should be given before operation.

When the lesion is high in the fundus, 8,000 to 10,000 gamma roentgens of radium are delivered to the uterine cavity. Schmidt uses doses of 20,000 to 30,000 gamma roentgens to the endometrial surface. When the lesion is low in the uterine cavity or has involved the cervical canal, a cancerocidal dose of radium should be delivered to the cervix and to points A and B.

Heyman's capsules or intrauterine tandems are employed according to one's choice. At the Radiumhemmet the uterus was perforated 27 times in 617 cases and on one occasion the irradiator broke and was lost in the peritoneal cavity.

My preference, for the normal size and large uterus, is the Schmidt Y applicator loaded with radium in both arms. It is easy and simple to use and the dose calculation is almost as simple as that of the single tandem. The Schmidt applicator, properly loaded, permits easy, adequate irradiation of the fundus uteri without danger of radium dislocation. For the small uterus a plastic, reflex, single tandem is preferred. It conforms, at body temperature, to the contour of the uterine cavity and can be sutured to the cervix to prevent dislocation.

If the disease involves the cervical canal and when the uterus is large enough, I used the Y applicator, then a week later I give additional radium to points A and B in a vaginal applicator with fixed ovoids and loaded with 20 or 25 mg. capsules. When the uterus is small and the disease involves the endocervix the same vaginal applicator is used with an added central tandem the length of which depends on the depth of the uterine cavity. Once the lesion has involved the cervical canal, its methods of spread is characteristic of that of cervical cancer and, since it is an adenocarcinoma, it should be irradiated accordingly. When the lesion is confined to the fundus, the treatment of choice is total abdominal hysterectomy followed six weeks later by bilateral salpingo-oophorectomy.

It is unnecessary to suture the cervix since the uterus is sterilized by irradiation in 75 per cent of cases (Schmidt), and in those cases where it is not sterilized the capacity of the cancer cells for implantation is tremendously decreased. It is accepted opinion that vaginal recurrences are from cancer cells squeezed through the blood vessels and lymphatics into the cellular tissues and fascial planes surrounding the vagina rather than from cells on the mucosal surfaces.

Postoperative radiation depends on the depth of myometrial invasion. When it is superficial the vagina alone is irradiated and with radium. When it is deep or has reached the serosal surfaces or the ovaries the entire pelvis is irradiated with cobalt-60 to tolerance—3,500 to 4,000 roentgens. When the lesion involves the cervical canal, in addition to hysterectomy and salpingo-oophorectomy, pelvic lymphadenectomy is indicated, since in this case the spread is as in cervical carcinoma although the lesion is adenomacarcinoma. Adequate irradiation

should precede these surgical procedures. Full cancerocidal doses, or none at all, should be used.

Surgery in the management of cervical carcinoma should be confined to carcinoma in situ, radioresistant lesions, Stages I and II lesions complicating complete uterine prolapse with large cystocele and rectocele, and to irradiation failures involving bladder, rectum or ureters.

Most important in the management of cancer of the cervix or corpus is the initial planning of therapy. Many failures are from lack of delivering cancerocidal doses of irradiation to the malignant lesions. Many such patients are permitted to progress beyond the point of salvage and end with mutilating and useless operations—all from lack of knowledge of the cancerocidal dosage of irradiation.

How often we hear attempts to justify mutilating surgical procedures! "She was a radiation failure—she had radium for 90 hours and x-ray therapy twice—two courses," which means absolutely nothing. We must know dosages in gamma roentgens reaching the lesion and points C, A, B and W. Calculating these dosages should be as commonplace as calculating amounts of drugs in a prescription. Upon completion of the course of radium therapy it is the gynecologist's responsibility to review the dosages with the radiologist in order that he can take up intelligently where the gynecologist left off, so that the patient will eventually receive cancerocidal doses of irradiation to the lesion at its origin and at its sites of spread.

It is most important that initial irradiation be adequate, because many patients develop resistance to subsequent irradiation just as resistance develops to chemotherapeutic drugs and to antibiotics.

Kottmeier recently stated that despite reports of good results from radical hysterectomy in selected cases the cervical carcinoma, the permanent results of primary adequate radiation in unselected series of cases are superior to those obtained by surgical treatment. Therefore, irradiation treatment should be the primary treatment in all cases of invasive cervical carcinoma.

Summary

The cervix, fundus and para-cervical tissues tolerate high doses of radiation. The primary lesion of carcinoma of the cervix is usually radiosensitive as are its metastatic lesions. This combination enables us to control a high proportion of carcinoma of the cervix. There is good evidence that lymph node metastasis can be controlled by irradiation in a large proportion of patients. The exact percentage has not been established.

The type of irradiation must be planned "Moss" depending on the clinical extent of the disease and associated circumstances, as pregnancy, cervical stump, narrow or large vagina. No single method is best in all circumstances. When radium is to be used, the Manchester modification of the Paris technique seems preferable. It is simple, easily learned and flexible. It is possible to get good results with other techniques when they are applied with a knowledge of the factors involved.

External irradiation is our most important single technique in the control of lesions in the advanced stages. It is the most practical and effective way of bringing the dose of radiation in the lateral part of the pelvis, to the desired level.

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(Continued on Page 428)

Fibrinolysis, frequently a complication of carcinoma of the prostate, has been described only occasionally as a complication of benign prostatic hypertrophy. A simple test of recognition is described as well as the treatment of this disorder.

Fibrinolysis in Benign Prostatic Hypertrophy*

LUCIEN C. KAVAN, M.D.
ANGEL BETTAGLIO, M.D.
HUGERT G. BUEHLER, M.D.

A 1942 graduate of the Harvard Medical School, Lucien C. Kavan, M.D., limits his practice to his specialty, urology. He is eligible for certification by the American Board of Urology.

Doctor Kavan is an Instructor in Urology at the University of Oklahoma School of Medicine.

At the time this paper was written, Angel Bettaglio, M.D., was taking a residency in Urology at the University of Oklahoma Medical Center. He is now in private practice in San Salvador, El Salvador.

Hubert George Buehler, M.D., graduated from Vanderbilt University School of Medicine in 1957. He is now taking his second year residency in surgery at the University of Oklahoma School of Medicine.

THE FOLLOWING case report is of interest for three reasons:

1. The infrequency of reported cases of fibrinolysis following operation for benign prostatic hypertrophy,
2. A growing interest in fibrinogen deficiency as a causative factor in surgical bleeding and
3. The good response in this case to fairly simple and inexpensive treatment.

A 70-year old white male was admitted to the Veterans Administration Hospital on 10-12-59 with a history of lower urinary tract obstructive symptoms of several years duration. He had required periodic urethral dilatations for stricture. There was a history of chronic cough and exertional dyspnea. The family and past histories were non-contributory. Physical examination showed a well-developed, thin, white male whose only positive physical findings were rales in both lung bases.

The admission laboratory work showed the complete blood count, urinalysis and blood urea nitrogen to be within normal limits. Serology was negative. A chest film revealed pulmonary emphysema with no evidence of tumor.

Urethral dilatations failed to alleviate the obstructive symptoms. Panendoscopy showed the cause of the obstruction to be a hypertrophied median lobe. No other abnormalities were apparent in the bladder or the remaining portion of the urethra.

An uncomplicated transurethral resection of the prostate was done under spinal anesthesia on 10-23-59. At the end of the resection the wash water returned clear. Fifteen minutes later the bladder drainage be-

*This paper is from the Department of Urology, University of Oklahoma Hospitals, Oklahoma City, Oklahoma.

came bright red. Traction on the catheter failed to slow the hemorrhage. The patient was then returned to the operating room where the bladder was emptied of blood clots and a few small bleeding areas were fulgurated transurethrally. After the bleeding was apparently controlled the patient was returned to the recovery room. The bladder drainage which was almost clear on leaving the operating room soon became bright red. Three pints of whole blood were given the patient while in the operating room and in the recovery room. Traction was again applied to the catheter.

Laboratory studies at this time showed a bleeding time of 35 seconds, a coagulation time of 9 minutes (Lee-White method), a prothrombin time of 13 seconds, a normal platelet count and a borderline fibrinogen level. A few centimeters of the patient's blood was allowed to clot in a test tube taped to the bed for observation.

Fresh whole blood was administered. The bleeding continued and two more pints of blood were required for replacement. After two hours the blood which had been allowed to clot in the test tube was still solid but gentle shaking resulted in complete and permanent dissolution of the clot. Two grams of fibrinogen were then given without any remarkable effect.

In spite of the fact the prostate was clinically benign it was decided to treat the patient as having a prostatic carcinoma. An occult or undetected carcinoma of the prostate might be responsible for the fibrinolysis which was occurring. We were cognizant of the rare possibility of fibrinolysis occurring in benign hyperplasia. Two hundred and fifty milligrams of diethylstillbesterol diphosphate were given intravenously. The bladder drainage began clearing shortly thereafter in a most dramatic and definite fashion. The same dosage of diethylstillbesterol diphosphate was repeated in six hours. The remainder of the post-operative course was uneventful.

Blood studies before discharge from the hospital were all within normal limits. There was a firm clot present in a blood sample which did not dissolve on shaking. No evidence of bony metastases was present on chest and abdominal x-rays. The acid phosphatase level was within normal limits. The pathologist's report was "hyperplasia of the prostate, glandular and fibromuscular elements predominating."

Comment

A case of post-operative hemorrhage due to fibrinolysis following transurethral resection of the prostate has been described. The initial impression was hemorrhage from open sinuses in the prostatic capsule. After traction on the catheter failed to control the bleeding a search was made for arterial bleeding. No active arterial bleeding was found on endoscopic examination of the prostatic capsule. The prothrombin time, bleeding and clotting time taken during the period of active bleeding were normal. The fibrinogen level was reported as borderline. Blood allowed to clot in a test tube retracted normally but the clot completely dissolved when agitated. On the basis of this simple test the diagnosis of fibrinolysis was made.

Although many cases of fibrinolysis in patients with carcinoma of the prostate are reported, only a few cases are mentioned in which fibrinolysis has occurred in operation for benign prostatic disease.

Estrogen therapy has been reported to inhibit fibrinolysis.[1] Cases have been reported, however, in which fibrinolysis has occurred while the patient was receiving estrogens.[2] Perhaps this discrepency is due to the dosage of estrogens given the two groups of patients.

We found the administration of large doses of diethylstillbesterol diphosphate had a dramatic effect in altering the fibrinolytic activity. There was a prompt cessation of bleeding and the patient's blood remained clotted following estrogen administration.

It is conceivable that many instances of excessive bleeding following prostate surgery may be the result of fibrinolytic activity. Confirmation may be obtained by merely observing a clotted specimen of blood. This test is simple and can become routine in unexplained post-prostatectomy bleeding. The administration of large doses of estro-

gen may promptly reverse the fibrinolytic activity. The use of estrogens in contrast to fibrinogen does not carry the hazard of intravascular thrombosis.

Summary

Post-operative bleeding due to fibrinolysis was seen in a patient with benign prostatic hypertrophy. Laboratory studies, including the fibrinolysin level, clotting and bleeding time, prothrombin time were all equivocal or normal and of little help in establishing the diagnosis. Dissolution of the clot in a sample of the patient's blood was most helpful in confirming our impression of fibrinolysis. The hemorrhage stopped after the intravenous administration of diethylstillbesterol diphosphate. The patient had previously received two grams of fibrinogen, Vitamin K, and fresh whole blood without any appreciable change in the blood loss.

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625 N.W. 10th, Oklahoma City, Oklahoma

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522 N.W. 13th, Oklahoma City, Oklahoma

ARISTOCRACY, *n.* Government by the best men. (In this sense of the word is obsolete; so is that kind of government.) Fellows that wear downy hats and clean shirts —guilty of education and suspected bank accounts.

From the Devil's Dictionary by Ambrose Bierce
—Sagamore Press, Inc.

MEDICAL CENTER

Message from the Dean

Much has been published in recent months about the dearth of qualified applicants for the nation's medical schools. For some schools, the present number of candidates is insufficient to assure an adequate influx of capable students.

The supply of physicians at any given time depends primarily on the number of students graduated. In 1959 the medical schools graduated 6,900 men and women. The association of American Medical Colleges has estimated that there will soon be 7,400 graduates annually. But, projecting the population trend, it is predicted that by 1975 we will need an annual graduation rate of 11,000.

It is obvious that the number and quality of applicants for medical school entrance is basic to the problem.

At the University of Oklahoma School of Medicine, we have seen a decline in the number of applications, just as other schools have. Nevertheless, with the exception of one year, each entering class has been filled.

We are limited largely to applicants from Oklahoma since the present University Regents' rule is that no more than ten percent of the class shall be selected from non-resident applicants.

The prospective student must have completed a minimum of three years in college. He must have at least a C plus average. The National Medical College Admissions Test is required, as is a letter of appraisal from his college. If the applicant meets these minimal requirements he is interviewed by the Board of Admissions, composed of nine faculty members. It is this group that accepts or rejects applicants.

The Board begins receiving applications for the next class in September of the preceding year. Interviews are scheduled at intervals over a three months period, and selection of the new entering class is completed in March.

This year, we had a gratifying increase in both the number and qualifications of applicants. Of 190 students who applied for admission, 150 met the minimal grade point requirement and 80 of these had an average of "B" or better. A total of 104 students and 18 alternates was accepted for the first year class which will enroll this fall. The four additional students (over the usual 100 beginning students per class) were accepted to allow for inevitable drop-outs.

Although the number of applicants and the number of students in an entering class represent interesting statistics, the crucial point is the number that finish the four year program and receive their degree of Doctor of Medicine. The quality of the applicants, and the selection, cannot evade this latter critical matter. Each physician should be a committee of one to encourage really able students in his community to seriously consider medicine as a career.

Mark R Everett

The etiology is a vague extra articular rheumatic disorder. The exact pathology is very vague and methods of treatment are many. This is a comprehensive and statistical review of 318 cases treated by surgery and conservative measures at the Bone and Joint Hospital, Oklahoma City, from 1939 to 1959.

Tennis Elbow*

CAPTAIN CHARLES A. ROCKWOOD, USAF (MC)

Captain Charles A. Rockwood, Jr., USAF (MC) graduated from the University of Oklahoma School of Medicine in 1956. A Junior Member of the International College of Surgeons, Captain Rockwood, in his third year of orthopedic residency, is serving an Air Force sponsored civilian residency at the University of Oklahoma Medical Center in the Department of Orthopedic and Fracture Surgery

TENNIS ELBOW is a syndrome characterized by chronic pain in the vicinity of the radiohumeral joint. Synonyms of tennis elbow are: epicondylitis, Rider's elbow, radiohumeral bursitis, epicondylalgia.

Etiology

The exact cause of tennis elbow is unknown. There are many proposed etiologies and these are listed in Figure I. Many authors think that tennis elbow is an occupational disease in which the forearm is supinated and pronated while the hand is forcefully gripping an object.

There are many ideas as to the exact location of pain, each depending on the etiology proposed. The majority think that pain on the tip of the epicondyle is the most common. Other sites of pain are the anterior or posterior surfaces of the epicondyle, the radiohumeral joint, and just above the epicondyle.

It is interesting to note that sporting activities made up only eight of the 318 cases reviewed, or 2%. Tennis was the sport blamed in three cases, or 1%. Water skiing, bowling, baseball, and golf were responsible in the other five.

Sex Incidence, Age Predominance and Work Classification

Of the 318 cases of tennis elbow there were 222 males, or 70%. Forty-four per cent of the patients were between forty to fifty years of age. Other age groups are shown below:

		Average Age Groups
14-16 years	=	1
25-30 years	=	38
30-40 years	=	62
40-50 years	=	140
50-60 years	=	63
60-70 years	=	14

In 53% there was a definite history of trauma to the elbow. In 47% the onset was insidious. This does not mean that in 53% of the cases the patient came immediately to the hospital following trauma but simply that he remembered trauma to the elbow in the past. The average duration of symptoms prior to the first examination in the clinic is seen in Figure 2.

*This paper was read before the Oklahoma Rheumatism Society Faculty Club, Oklahoma City, Oklahoma, January 13, 1960. The contents of this article are the personal views of the Air Force author and are not to be construed as statement of official Air Force policy.

FIGURE NO. I

ETIOLOGY

1. Traumatic periostitis.
2. Arthritis, synovitis, sprain, adhesions or torn capsule of the radiohumeral joint.
3. Arthritis, synovitis, sprain or adhesions of radio-ulnar joint.
4. Displaced, frayed, torn or inflamed orbicular ligament.
5. Sprained, torn radial-collateral ligament.
6. Inflamed or calcified radiohumeral bursa, (Osgood's bursa).
7. Inflamed or calcified subcutaneous epicondylar bursa.
8. Nipped synovial fringe within radiohumeral or radio-ulnar joints.
9. Tear or fibrositis of extensor origin.
10. Tear or fibrositis of supinator-brevis.
11. Torn pronator teres.
12. Torn extensor carpi-radialis-longus.
13. Torn extensor carpi-radialis-brevis.
14. Tear or fibrositis of branchioradialis.
15. Tear, sprain, fibrositis of extensor-digitorum-communis.
16. Myositis or tear of extensor muscles.
17. Torn anconeus.
18. Radial incongruence.
19. Twist of the whole radius.
20. Rheumatism, gout, influenzal sequelae, focal sepsis, arthritic diathesis.
21. Neuritis or radial posterior interosseous, or cutaneous antibrachii lateralis.
22. Saturnism.
23. Osteomalacia.
24. Deposits about olecranon.
25. Periostalgia.
26. Osteochondritis.

FIGURE NO. III

CASES SHOWING POSITIVE X-RAY CHANGES

1. Calcified deposits about joint	12
2. Soft tissue density	2
3. Osteoporotic bone about joint	1
4. Loose bone fragments about joint	6
5. Congenital deformity about joint	3
6. Large bony prominence about joint	4
7. Degenerative changes about joint (Spurs, irregularities and roughening)	25
	53 (17%)

FIGURE NO. II

AVERAGE DURATION OF SYMPTOMS PRIOR TO FIRST EXAMINATION AT THIS CLINIC

	Number of Patients
Acute	24
1 week	4
2 weeks	35
3 weeks	12
4 weeks	47
5 weeks	3
2 months	55
3 months	43
4 months	20
5 months	5
6 months	29
7 months	4
8 months	7
9 months	3
One year	10
Two years	14
Three years	2
Five years	1
Total	318

FIGURE NO. IV

1. Rest
2. Strapping
3. Pressure pads
4. Painting with Iodine, etc.
5. Blistering
6. Hot baths
7. Hot air
8. Luminous heat
9. Galvanism
10. Faradism
11. Diathermy
12. Carbolic acid
13. Novocain
14. Removal of focal sepsis
15. Vitamin D
16. Thyroid gland
17. Salicylates, given orally
18. Massage
19. Cock-up splintage
20. Mobilization
21. Periosteal excision
22. Division of the muscular origin at the epicondyle
23. Bursting or removing bursa
24. Disengaging or removing synovial fringes
25. Excision of orbicular ligament
26. Fasciotomy of common extensor muscle belly

FIGURE NO. V

A REVIEW OF SURGICAL TREATMENT IN CASES OF TENNIS ELBOW: B & J HOSPITAL

Type Occupation	Injury	Conservative Treatment	SURGERY	Results
Bindery Worker (Seen 2 mo. post-injury)	Bumped elbow	Physical Therapy, splinting, injections, with no relief	Partial stripping + excision of extensor mechanism	Not relieved at the end of 2 yrs. POOR
Manual Laborer (Seen 2 days post-injury)	Bumped elbow	Physical Therapy, rest in sling—No relief	Exploration + tenotomy, extensor fascia	Not relieved. Further surgery recommended but declined. POOR
Oil Field Laborer (Seen 1 mo. post-injury)	Bumped elbow	Rest in sling, Physical Therapy, Injections—No relief	#1. Extensor fasciotomy #2. Excision of external condyle and partial synovectomy, radial head	Not relieved at 6½ mo. post-injury. Complete relief following 2nd operation. GOOD
Driller (Seen 5 mo. post-injury)	Bumped elbow	Injections, Physical Therapy, rest in sling, No relief	Periosteal stripping + partial resection of lateral epicondyle	Returned to work at 8 mo. post-injury with complete relief. GOOD.
Roughneck, Oil Fields. (Seen 6 mo. post-injury)	Fall onto extended arm	Injections, Physical Therapy + rest—No relief	Partial excision of lateral condyle	Returned to work at 8 mo. post-injury with complete relief. GOOD
Driller (Seen 3 mo. post-injury)	Bumped elbow	Injections, Physical Therapy and rest—No relief	Fasciotomy, extensor group	Returned to work at 8 mo. post-injury with complete relief. GOOD
		(Same patient with 2nd injury, opposite arm)		
(Seen 9 da. post-injury)	Struck on elbow by pipe	Injections, rest, Physical Therapy—No relief	Excision of olecranon bursa	Returned to work at 2 mo. post-injury with complete relief. GOOD
Engineer (Seen 7 da. post-injury)	Bumped elbow	Injections, Physical Therapy—continued at work—No relief	Excision of calcified mass from common extensor at lateral condyle	Returned to work at 7 da. post-op with complete relief. GOOD
Miller (Seen 1 mo. post-injury)	Struck on elbow by a pipe	Injections, rest in sling—No relief	Excision of synovial bursa	Returned to work at 3 mo. post-injury with complete relief. GOOD
Construction Laborer (Seen 1 mo. post-injury)	Slip and fall onto elbow	Deep X-ray Therapy, Physical Therapy, rest in sling—No relief	Stripping of lateral condyle	Returned to work at 2 mo. post-injury with complete relief. GOOD
Aircraft Flight Test Mechanic (Seen 12 da. post-injury)	Struck elbow on an air-craft	Deep X-ray Therapy, Physical Therapy, Cast + injections—No relief	Exploration and excision of fibrous tissue from inferior lateral condyle	Returned to work at 7 mo. post-injury with complete relief. GOOD
Petroleum Still-man (Seen 7 mo. post-injury)	Bumped elbow	None here—Surgery recommended—No relief on conservative treatment elsewhere	Exploration and excision of bursa	Returned to work at 7½ mo. post-injury with complete relief. GOOD

FIGURE NO. VI
SUMMARY OF YEARLY TREATMENT TYPES

Year	Surg.	Gluco-steroids	Novocain Inj.	Comp. F	Butazol-idin	Manip. Cast	Thoraz-ine	Deep X-Ray	P.T.	Splint	Vitam.	Hor-mones
1945	0	0	7	0	0	1	0	2	0	0	0	0
1946	0	0	6	0	0	0	0	4	0	0	0	0
1947	0	0	0	0	0	0	0	0	1	0	0	0
1948	0	0	11	0	0	0	1	2	1	0	0	0
1949	1	0	11	0	0	0	1	2	1	0	0	1
1950	0	0	8	0	0	0	0	1	2	1	0	1
1951	1	1	12	0	0	0	0	3	0	0	0	0
1952	3	6	11	2	0	0	0	1	1	1	1	1
1953	2	6	1	0	1	0	0	0	0	0	0	0
1954	0	11	1	9	1	0	1	0	1	1	0	2
1955	2	28	0	0	6	0	0	1	3	1	0	1
1956	1	17	0	13	2	0	0	1	0	0	1	0
1957	1	15	0	3	3	0	0	0	3	0	0	0
1958	0	27	0	1	2	1	0	0	1	0	0	0
1959	0	34	0	3	0	4	0	0	4	0	0	0

Manual laborers, such as drillers, welders, and truck drivers made up 170 cases, or 56%. People involved in lighter types of occupations such as nursing, teaching, and waiting tables made up 100 cases, or 31%. The remaining 13% were housewives whose work might be of either type.

Clinical Picture

Irrespective of whether there is a history of trauma, the painful syndrome of tennis elbow comes on gradually. It is characterized by dull aching about the lateral aspect of the elbow joint but becomes a very sharp pain, radiating down the forearm with a grasping action of the hand and rotation of the forearm. Grasping requires a setting of the common extensor group on the lateral epicondyle and any rotation of the forearm places stress on this extensor group. The pain is so severe that even small objects, such as a teacup, may be dropped. Generally there is no swelling or discoloration about the lateral elbow region. As has been mentioned, the most common sites of pain are over the tip of the lateral epicondyle of the humerus, and pressure here reproduces the sharp and intensified pain. X-ray changes in tennis elbow are uncommon. In this series x-ray changes were found in 53 cases, or 17%. These changes are shown in Figure 3.

The right arm was involved in 207, or 62%. The left arm was involved in 93, or 29%. Symptoms were bilateral in 18, or 7%.

From 1939 to the present time, at the Bone and Joint Hospital and McBride Clinic 307, or 97% of the patients, were treated by conservative measures. Eleven patients were treated by surgical means. Types of treatment reported in the literature include a wide variety of measures (see Figure 4).

Surgical Treatment

Of the eleven patients with tennis elbow treated surgically, two had poor results. It is of interest to note that all eleven of these were industrial, all were manual laborers and all had definite history of trauma to the elbow. All patients were treated with conservative measures for a period of at least six months without relief before surgical means were employed.

Different operative procedures were used. These included fasciotomy of the common extensor muscle belly; partial excision of the epicondyle; periosteal stripping; excision of the bursa, and various combinations of these procedures.

The average amount of time lost before work could be resumed was five months. (Refer to Figure 5 for summary of surgically treated patients.)

FIGURE NO. VII

NUMBER OF TREATMENTS AND RESULTS

Year	TREATMENT	No. of cases	NO. OF TREATMENTS REQUIRED						RESULTS	
			1	2	3	4	5	Other	Complete Relief	No Relief
1939 to 1945	No specific = Rest at home, home Physical Therapy, etc.									
1945	Surgery	0								
	Manip. + sling	1	1	0	0	0	0	0	1	0
	Inj. Novocain	7	5	2	0	0	0	0	7	0
	Physical Therapy	2	0	0	0	0	0	1 month	2	0
	Total	10							10	0
1946	Surgery	0								
	Inj. Novocain	6	4	2	0	0	0	0	6	0
	Deep X-Ray Therap.	4	0	0	0	0	0	3 weeks	4	0
	Total	10							10	0
1947	Surgery	0								
	Physical Therap.	1	0	0	0	0	0	1 month	1	0
	Total	1							1	0
1948	Surgery	0								
	Deep X-Ray Therap.	2	0	0	0	0	0	3 weeks	2	0
	Cast	1	1	0	0	0	0	0	1	0
	Inj. Novocain	11	9	2	0	0	0	0	11	0
	Physical Therap.	1	0	0	0	0	0	1 month	1	0
	Total	15							15	0
1949	Surgery	1							1	0
	Inj. Novocain	11	9	2	0	0	0	0	9	2
	Deep X-Ray Therap.	1	0	0	0	0	0	3 treat.	1	0
	Oral = Dolophine + Tolserol	1	0	0	0	0	0		Unknown-seen only once	
	Oral Hormone	3	0	0	0	0	0	1 = unknown	0	0
								1 = 7 mo.	1	0
								1 = 3 mo.	0	1
	Sling	1	0	0	0	0	0	1 month	0	1
	Vitamins	1	0	0	0	0	0	2 weeks	0	1
	Physical Therap.	1	0	0	0	0	0	1 month	1	0
	Cast	2	2	0	0	0	0		2	0
	Total	22							15	5
1950	Surgery	0								
	Inj. Novocain	8	5	2	1	0	0	0	8	0
	Cast	1	1	0	0	0	0	0	0	1
	Physical Therap.	2	0	0	0	0	0	1 = 1 mo.		
								1 = 2 wks.	2	0
	Oral Hormone	1	0	0	0	0	0	1 month	1	0
	Deep X-Ray Therap.	1	0	0	0	0	0	3 treat.	1	0
	Oral Prednisolone	1	0	0	0	0	0	3 wks.	1	0
	Total	14							13	1
1951	Surgery	1							1	0
	Inj. Hyaluronidase	1	1	0	0	0	0	0	1	0
	Inj. Novocain	12	8	3	1	0	0	0	11	1
	Deep X-Ray Therap.	3	0	0	0	0	0	1 = 3 treat.		
								1 = 5 treat.		
								1 = 6 treat.	3	0
	Total	17							17	0

1952	Surgery	3							3	0
	Inj. Hydrocortisone	8	5	3	0	0	0	0	8	0
	Inj. Novocain	10	8	1	0	0	0	1 = 6 inject.	9	1
	Deep X-Ray Therap.	1	0	0	0	0	0	3 treat.	1	0
	Oral Hormone	1	0	0	0	0	0	2 weeks	1	0
	Inj. B-12	1	0	0	0	0	0	31 shots	1	0
	Cast	1	1	0	0	0	0	0	1	0
	Inj. Wyadase	1	0	0	0	0	0	0	1	0
	Total	26							25	1
1953	Surgery	2							2	0
	Inj. Hydrocortisone	7	5	1	1	0	0	0	7	0
	Oral Butazolidin	1	0	0	0	0	0		1	0
	Total	10							10	0
1954	Surgery	0								
	Inj. Hydrocortisone	20	13	4	2	0	1	0	19	1
	Oral Thorazine	1	0	0	0	0	0		Unknown	
	Cast	1	1	0	0	0	0	0	1	0
	Inj. Novocain	1	1	0	0	0	0	0	1	0
	Oral Hormone	2	0	0	0	0	0	1 = 2 wks.		
								1 = 1 mo.	2	0
	Physical Therap.	1	0	0	0	0	0	2 wks.	1	0
	Butazolidin	1	0	0	0	0	0	2 wks.	1	0
	Total	27							25	1
1955	Surgery	2							2	0
	Oral Butazolidin	6	0	0	0	0	0	2 = 5 mo.		
								4 = 1 mo.	6	0
	Physical Therap.	3	0	0	0	0	0	1 mo.	3	0
	Inj. Hydrocortisone	27	15	8	4	0	0	0	26	1
	Oral Cortisone	1	0	0	0	0	0	0	Unknown	
	Splint	1	1	0	0	0	0	0	1	0
	Deep X-Ray Therap.	1	0	0	0	0	0	3 treat.	1	0
	Oral Hormone	1	0	0	0	0	0	3 mo.	1	0
	Total	42							40	1
1956	Surgery	1							0	1
	Inj. Hydrocortisone	30	16	8	4	2	0	0	29	1
	Oral Butazolidin	2	0	0	0	0	0	2 = 1 mo.	2	0
	Deep X-Ray Therap.	1	0	0	0	0	0	1 treat.	1	0
	Splint	1	1	0	0	0	0	0	1	0
	Total	35							33	2
1957	Surgery	1							0	1
	Physical Therap.	3	0	0	0	0	0	1 = 2 treat.		
								2 = 1 wk.	3	0
	Inj. Hydeltrasol	1	0	0	0	0	0	0	1	0
	Inj. Hydrocortisone	17	16	1	0	0	0	0	17	0
	Oral Butazolidin	3	0	0	0	0	0	1 mo.	2	1
	Total	25							23	2
1958	Surgery	0								
	Inj. Hydrocortisone	23	16	6	1	0	0	0	22	1
	Inj. Prednisolone	5	4	1	0	0	0	0	4	1
	Oral Butazolidin	2	0	0	0	0	0	1 month	2	0
	Manip. + Cast	1	0	0	0	0	0	0	1	0
	Physical Therapy	1	0	0	0	0	0	12 da.	1	0
	Total	32							30	2
1959	Surgery	0								
	Inj. Hydrocortisone	32	23	6	1	2	0	0	30	2
	Inj. Prednisolone	2	1	0	0	0	0	0	2	0
	Inj. Hydeltrasol	3	0	3	0	0	0	0	3	0
	Manip. + Cast	4	4	0	0	0	0	0	4	0
	Physical Therap.	4	0	0	0	0	0	3 to 4 da.	4	0
	Total	45							43	2

Conservative Treatment

Most authors think that with conservative measures relief can be obtained. From a review of the 307 cases treated without operation, we found that relief can be obtained with a variety of drugs, however, a more rapid recovery can be accomplished by injections of the newer glucocorticoids (Figure 6). This chart shows the evolution and the types of treatment up to the present time in the 318 cases reviewed from the Bone and Joint Hospital and McBride Clinic.

Figure 7 is a resume by year of the different types of treatment and the results of each. Relief of the pain was accomplished by use of such products as oral sex hormones, vitamins, Tolserol, and Thorazine.

Recurrence of tennis elbow after a "cure" has been discussed in the literature as a rarity. Current experience is against this as a principle. In this series 17, or 5%, of the 318 cases had recurrence. The time after the original cure varied from one to seven years. In three cases there were two recurrences.

Summary

1. The clinical picture of tennis elbow is that of persistent dull ache about the lateral epicondylar region of the elbow which is intensified to a sharp radicular pain down the forearm by grasping motions of the hand and rotation of the forearm.

2. The etiology is multiple but the exact cause is unknown.

3. Pain is most common over the tip of the lateral epicondyle of the humerus.

4. X-ray changes are uncommon.

5. Males are predominantly involved, 7 to 3.

6. The most common age bracket is from forty to fifty years.

7. Trauma is reported in about one-half of the cases.

8. The syndrome most commonly involves manual laborers. It is rare in athletes, and is particularly rare in tennis players.

9. The right arm is most commonly involved, however, in some patients biolateral involvement may be seen.

10. The majority of patients can be treated with conservative measures with excellent results.

Acknowledgment

I would like to express my sincere appreciation to Mrs. Grace Herndon, Doctors J. N. Owens, Jr., and Richard W. Payne for their help in preparing this paper.

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605 N.W. 10th, Oklahoma City, Oklahoma

ABSTRACTS

A Study of the Roentgen Appearance of Cranial Vault Sutures: Correlation With Their Anatomy

JOHN B. CHRISTENSEN, Ph.D.,* ERNEST LACHMAN, M.D.,** and ALICE M. BRUES, Ph.D.***

Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 83: 615, 1960

In addition to representing a pitfall in the diagnosis of skull fractures, the roentgen appearance of cranial vault sutures is of interest to the radiologist because: a) it furnishes important evidence in the diagnosis of abnormal acceleration or delay of sutural fusion; and b) it offers a means of evaluating physiologic maturation throughout life and at ages where other indicators of skeletal differentiation have lost their usefulness.

In order to test the reliability of the roentgen evaluation of the sutural status a comparative study was made which correlates the radiologic findings on the vault sutures of 26 intact cadaver heads with the results of direct observation on the external and internal aspects of the calvaria of the same heads after removal of all soft tissues. It was found if no suture line is seen on the roentgenogram, that the endocranial suture is at least three-fourths closed. A suture which is open anatomically on its outer and inner aspects is invariably demonstrable on the roentgenogram, either as an intermittently open line or more commonly as a completely open line. If the suture is closed endocranially, but open ectocranially, all variants of radiographic findings occur from disappearance of the suture line to complete patency. Frequently and surprisingly, intermittently or completely open radiographic suture lines were visible in skulls, in which both ecto- and endocranial surfaces were three-fourths to fully closed. Radiographically open suture lines seem to increase in frequency when calvaria with ecto- and endocranially open sutures are compared with crania displaying beginning endocranial fusion.

Statistically proved is the paradoxical increase in suture visibility when we compare skulls with endocranial fusion and ectocranial patency with skulls in which endocranial fusion is combined with beginning closure of the ectocranial aspect. It is suggested that this radiographic "opening-up" of the suture may be explained: a) by bone absorption in the diploe occurring with suture closure of the ecto- and endocranial surface aspects, or b) by the plane of the suture assuming a more favorable angle for visibility as the suture begins to close. Either of these possibilities would result in increasing visibility of the sutural gap in the diploe.

The dentations of a suture extend through the depth of the suture into the diploe and beyond. Even the dural aspect of the suture shows more serrations than is generally believed. It is suggested that the roentgenologic appearance of a suture is based on its total cross section and that it therefore may give a better indication of the sutural status for purposes of anthropological, physiological, and age evaluation than the anatomical appraisal of only its surface aspects.

*James Picker Foundation Fellow in Radiological Research 1958-1959; at present: Assistant Professor of Anatomy, George Washington University, Washington, D.C.
**Professor and Chairman, Department of Anatomy.
***Associate Professor, Department of Anatomy.

Variations in Endocrine Gland Function In Postpartum Pituitary Necrosis

CARL W. SMITH, JR.,* and R. PALMER HOWARD**
The Journal of Clinical Endocrinology and Metabolism 19: 1420-1436, Nov., 1959

Postpartum pituitary necrosis in 4 patients was manifest by classic panhypopituitarism with secondary insufficiency of the thyroid, adrenal cortex, and ovary. Three other patients presented atypical syndromes characterized by virtually normal thyroid function, variable degrees of adrenocortical insufficiency, and normal to slightly elevated urinary excretion of gonadotropin, with or without the resumption of menses. Low 24-hour thyroidal I^{131} uptakes were associated with low urinary gonadotropin levels, and normal uptakes with higher gonadotropin levels. It is inferred that in some cases of postpartum pituitary necrosis there may be decreased ACTH secretion, nearly normal TSH secretion, and restored gonadotropin secretion which, however, may not be adequate for normal ovarian function.

*Assistant Professor of Medicine.
**Associate Professor of Research Medicine, Cardiovascular Section of the Oklahoma Medical Research Foundation.

Effects of Parathyroid Extract (PTE) on Serum and Kidney Utilizing Sulfur-35

R. H. BRADFORD,* R. P. HOWARD,** W. JOEL,*** and M. R. SHETLAR.****
Fed. Proc. 18: 196, 1959

This laboratory has previously shown that large doses of PTE (Lilly & Co.) produce renal calcification and elevated serum glycoprotein and seromucoid. In studying the possible role of glycoproteins in the calcification process, we have utilized $Na_2S^{35}O_4$ (Dziewiatkowski, J. Exptl. Med. 93: 451, 1951). A control group of Holtzman rats were given only the sulfate; a second group was given the sulfate, then 250 units of PTE in 3 days. Paper electrophoresis of the individual serum samples from the PTE-treated animals revealed marked and significant decreases of both albumin protein and albumin glycoprotein fraction, while the globulin fractions were increased. There was almost twice the radioactivity, most nondialyzable, in the pooled sera of the PTE-treated animals as in the con-

trols. Continuous electrophoresis of these sera revealed that both had a radioactivity peak in the alpha globulin region. Kidney homogenates were fractionated by solubility techniques. The PTE-treated group had more radioactivity in almost all fractions; much of this was non-dialyzable. Subsequent perchloric acid treatment resulted in 2 sub-fractions from the PTE-treated group which, on paper electrophoretic analysis, had Alcian blue positive areas. Radioautographic study of kidney sections revealed localized areas of radioactivity over the calcified lesions in the PTE-treated group.

*Research Associate, Endocrinology Section, Oklahoma Medical Research Foundation.
**Associate Professor of Research Medicine; Endocrinology Section, OMRF.
***Professor of Pathology.
****Professor of Biochemistry.

Antagonistic Effects of Cortisone and Parathyroid Extract on Serum And Kidney Components

REAGAN H. BRADFORD,* R. PALMER HOWARD,**

WALTER JOEL,*** and M. R. SHETLAR.****

Program, Am. Chem. Soc., March, 1959

Parathyroid extract (PTE) has previously been shown to elevate serum calcium, glycoprotein, and seromucoid, as well as produce increased renal polysaccharide accompanied by calcification (Sheltar, M. R., **et al.**, Endocrinology, 59, 532 (1956). Cortisone, administered simultaneously with PTE, has been found to antagnoize the PTEeffects on serum calcium and renal calcification (Laron, Z., **et al.**, A.M.A. Arch. Path., 65, 125 (1958). In continuing our investigations of the calcification process utilizing PTE, we have determined the effect of cortisone on certain other aspects of the PTE-induced alterations in serum components and kidney which may be of importance in this process.

Three groups of Holtzman rats were injected twice daily for 3 days with either cortisone acetate (total of 30 mg.), PTE (total of 2.70 ml.), or PTE (2.70 ml.) and cortisone (30 mg.); a fourth received no treatment. All were sacrificed 72 hours after the first injection.

Chemical determinations on the individual serum samples revealed that the marked elevation of total serum glycoprotein produced by PTE was not obtained when cortisone and PTE were administered simultaneously. Further, the striking alteration in electrophoretic fractions of both serum protein and serum glycoprotein resulting after administration of PTE alone did not occur when it was administered with cortisone.

The histochemical study of the kidney sections utilizing the hematoxylin and eosin staining technique indicated a marked reduction in PTE-induced calcification when both PTE and cortisone were given, thus confirming the above-noted work of Laron, **et al.** We have further substantiated this observation by applying the Kossa staining technique which may be used for a more positive demonstration of calcium. Of similar interest to a study of the calcification process are our data showing that renal polysaccharide and acid mucopolysaccharide, markedly increased by PTE administration, are only moderately increased above normal when cortisone is simultaneously given.

*Research Associate, Endocrinology Section, Oklahoma Medical Research Foundation.
**Associate Professor of Research Medicine; Endocrinology Section, OMRF.
***Professor of Pathology.
****Professor of Biochemistry.

The Oral Yeast-Lactobacillus Relationship II. Yeast-Lactobacillus-Saliva Interrelationships

THOMAS E. WILSON* and PAUL W. GOAZ.**

Journal of Dental Research, 38: 1044-1051, Sept.-Oct., 1959

In a diphasic culture system, an oral strain of Candida albicans is an active donor of materials with pyridoxamine, calcium pantothenate, niacin, and riboflavin activity. The C. albicans did not supply a significant amount of folic acid, or any essential amino acid, except lysine. Pooled, whole saliva contained all of the vitamins, and eight of the eleven amino acids essential for the growth of lactobacillus. In the presence of saliva, the syntrophic effect of the yeast, attributed to the release of vitamins, was minimized, due to availability of these same factors in the saliva.

*Department of Bacteriology, Southwestern Louisiana Institute, Lafayette, Louisiana.
**Assistant Professor in Research Dental Surgery.

The Pharmacology of Placebos

STEWART WOLF.*

Pharmacological Reviews, 41: 689-704, Dec. 1959

It is important to realize that every contact with a physician may be a meaningful situation to the patient, and, hence, may set off visceral responses which can have salutary or adverse effects on his health and well-being. A placebo in the form of a drug may potentiate such effects. So may a host of rituals and procedures including surgery and a variety of forms of psychotherapy. To establish the intrinsic worth of any therapeutic maneuver, a well-designed and properly controlled test situation is required. The therapeutic experiment requires skill, experience and sophistication. Until recently, however, few highly qualified investigators had caught its challenge. It is hoped that their efforts will supplant those of the legions of amateur investigators who, year after year, convert the free pills of the detail men into enthusiastic print. Pickering has referred to the controlled therapeautic trial as the most important medical development of the past ten years (49). Recognition of the power and the properties of the placebo has been an important part of this development and the placebo, for an understanding of the extensive bodily changes it may induce, has gained a more dignified place in the therapeutic armamentarium.

*Professor of Medicine, Consultant Professor of Psychiatry, Neurology and Behavioral Sciences.

John W. Kelly, Ph.D., Receives Guggenheim Fellowship

John W. Kelly, Ph.D., associate professor of anatomy, has received a $3,600 Guggenheim Foundation fellowship in support of his plans to do cell research at the Medical Nobel Institute of the Karolinska Institutet in Stockholm.

He will resign from the University of Oklahoma School of Medicine faculty this summer to go to Sweden for a three-year stay at the Nobel Institute, where he will study the quantitative cytochemistry of nucleoprotein synthesis in normal and neoplastic cells.

Doctor Kelly came here last fall from Medical College of Virginia. This is his second Guggenheim award and his second appointment to the Institute. He was a Guggenheim guest investigator there in 1957-58.

Alpha Omega Alpha Inducts Seventeen

Alpha Omega Alpha inducted 17 men April 19 at the annual initiation banquet at the University of Oklahoma Medical Center. Speaker was Robert H. Ebert, M.D., director of Medicine at University Hospitals of Cleveland and Western Reserve University, who also gave the medical honorary's spring lecture for students and faculty.

Initiates included three faculty members, Gilbert S. Campbell, M.D., Ph.D., professor of surgery, Harold G. Muchmore, M.D., assistant professor of Medicine, and Louis J. West, M.D., professor and head of the Department of Psychiatry.

Students inducted: Edward N. Brandt Jr., Marietta; Timothy Hardin Dennehy, Tulsa; Bobby Gene Eaton, Weatherford; Paul Edwin Kaldahl, Oaks; Herbert Harold Lenabaugh, Hinton; Leo Meece, Frederick; Warren F. Ricchetti, Konawa, and Philip Judson Campbell, Donald C. Gilliland, William Mott Johnson, and Paul Albert Leap, Oklahoma City, graduating seniors; Robert F. Bell, Seminole; Victor L. Robards, Tahlequah, and Jane Self, Tulsa, juniors.

Marvin R. Shetlar, Ph.D., Appointed Professor of Biochemistry

Marvin R. Shetlar, Ph.D., a faculty member since 1948, was promoted from associate professor to professor of biochemistry in recent action of University of Oklahoma Regents.

At the same time, one addition to the part-time faculty was approved. Thomas E. Nix Jr., M.D., a 1952 graduate of Northwestern University Medical School, was appointed junior clinical assistant in dermatology. He is a former instructor in the Department of Medicine at Tulane University Medical Center.

Phillip C. Johnson, M.D., Leaving

Philip C. Johnson, M.D., associate professor of medicine and chief of radioisotope service at the Oklahoma City VA Hospital, will leave the Medical Center July 1 to join the faculty of Baylor University College of Medicine, Houston.

Doctor Johnson has been appointed associate professor of medicine at Baylor and will direct the radioisotope laboratory at Methodist Hospital. He came to the OU School of Medicine in 1955; developed the radioisotope service at VA.

Special Article

Factors Relating To Physician Distribution in Oklahoma 1950-1959

Part IV of a Series**

ROBERT C. LOWE, M.D.*
JAMES A. HAGANS, M.D.*
PEARL FISHER, Ph.D.*

Before the relationship between physician distribution and hospital construction and operation can be studied further, it will be necessary to deal with the question brought up earlier in Part II regarding the influence of physician concentration on physician migration. The initial determinants of physician concentration in a free enterprise system are simultaneously the determinants of their distribution. Some of these factors have been dealt with previously.

In contrast to the usual direct relationship between population concentration and population organization, referred to at the beginning of Part II, an inverse relationship may be observed in situations of relative isolation. Particularly, in situations of isolation, it is recognized that the best use of available skills and manpower are realized through organization. The wagon train and the frontier fort were early examples of this, and illustrate not only the phenomenon of organization but also its subphenomenon of division of labor.

Group medical practice is a particular form of concentration and organization for making medical care available at the "operating level" (speaking in the functional rather than the surgical vernacular!). This form of practice became known to the public early through a spreading knowledge of major "clinic" organizations in various parts of the country. The initiation and growth of clinics has been a slow and limited process. Its development has, however, demonstrated a variability consistent with that of other free enterprise endeavours in the United States, and has also served, especially in the larger groups, to offset for the public the disadvantages of dispersal of skills brought about by specilization.***

*All three of the authors are with the Department of Preventive Medicine and Public Health, University of Oklahoma School of Medicine; Doctors Hagans and Fisher are also with the Biostatistical Unit of the Department of P.M. and P.H.: in addition, Doctors Lowe and Hagans serve with the Department of Medicine.

**Part III appeared in the April 1960 issue of this Journal.

***In a recent study of medical education (The Student-Physician; ed. Morton, R. K. et al, Harvard U. Press. 1957) an appendix of several pages is devoted to a semantic disclaimer of Socialism. In a study of present day medical education for practice, vis a vis the variety of need situations, coming out of a setting which emphasizes comprehensive care and teaching, it is startling to find no mention of group practice, to which that setting has been compared. The authors of the present series have taken for granted that there is no need to prove the obvious: that there is no inherent connection of group practice with any political or economic philosophy.

Migration and Replacement of M.D.'s in Communities with and without Group Practice; 1950-1959

Community Size	(% range) Avg. & % M.D.'s in Gr. Pr.	Col. 1 % M.D.'s remaining	Col. 2 % M.D.'s New	Col. 3 % total change	Col. 4 % M.D.'s replaced	Col. 5 1959 % ‹ 60	Col. 6 % change ‹ 60	Col. 7 % change › 60
Section								
A. No group practice:								
1500-6000	0.0	47.3	38.7	—15.6	63.2	72.3	— 1.6	—41.5
6000-10,000	0.0	60.4	33.3	— 9.4	76.2	64.6	— 3.1	—19.0
10,000-20,000	0.0	66.7	26.0	— 9.9	70.4	73.2	0.0	—29.6
20,000-38,000	0.0	72.2	37.3	+13.4	151.8	80.9	+27.1	—22.2
Section								
B. Group Practice:								
	(50-100)							
1500-6000	78.1	51.4	43.8	— 8.6	82.4	90.6	+38.1	—64.3
	(44.4-83.3)							
6000-10,000	57.1	51.2	37.8	—14.0	66.7	78.4	— 6.5	—33.3
	(40.0-53.3)							
10,000-20,000(1)	45.8	62.9	20.2	—20.0	43.6	73.8	—10.1	—37.0
	(17.0-31.1)							
10,000-20,000(2)	26.1	58.4	42.7	0.0	100.0	72.0	+12.5	—27.6
	(6.6-29.2)							
20,000-38,000	15.8	71.5	39.5	+12.0	140.0	73.4	+25.0	—13.0

Section
C. Net Change in No. of M.D.'s (from Section B):

Community Size	Individual Practice	Group Practice	% of Est. Loss* Offset by Gr. Pr.
1500-6000	—12	+13	75.0
6000-10,000	—15	+10	47.6
10,000-20,000(1)	—27	+12	39.1
10,000-20,000(2)	—14	+10	20.0
20,000-38,000	+8	+15	167.0

*Estimated loss from 1950 level calculated from all M.D.'s in same proportion as loss (or gain) for those in individual practice in 1950.

TABLE VII

No one objectively doubts that the medical service pattern in small communities and farm populations is changing. Individual medical practice is decreasing in these areas for justifiable and well recognized reasons. This would be of no real concern to these segments of the population nor to the medical profession if the need was always elective and/or travel time to the nearest large community was always such as to encourage a high and early standard of use of preventive, diagnostic and treatment services available from the physicians practicing in the larger centers.

It should also be recognized by the public, and of course it is not, that stability in its supply of good local medical care depends upon its use of it. A reviewer's note quoted in a 1950 study of Belcher (2) on service utilization by farmers states that,

> "For example, it has been found that in certain areas rural families will travel great distances for a doctor and medical care, even though the same quality of service may be available near by. On the other hand, in areas sometimes not more than 100 miles distant, rural families are taking advantage (sic) of the nearest doctor and medical services."

This is an important aspect of the problem. At first thought it suggests that the long distance search is determined by non-objective relative status assigned by the public to the sources from which medical services may be sought. It requires further study.

Analysis of Table VII by Number of M.D.'s in Group Practice and Individual Practice, According to Community Size; 1950-1959

Section 1

Community Size	Col. 1 No. of towns	1950 Col. 2 No. M.D.'s in Ind. Pr.	1950 Col. 3 No. M.D.'s in Gr. Pr.	1959 Col. 4 No. M.D.'s in Ind. Pr.	1959 Col. 5 No. M.D.'s in Gr. Pr.	Col. 6 % of all M.D.'s in Gr. Pr. 1950	Col. 7 1959
1500-6000	50	233	18	189	31	7.2	14.8
6000-10,000	9	91	12	68	22	11.7	24.4
10,000-20,000	12	224	36	170	58	14.4	25.4
20,000-38,000	8	220	15	241	30	6.4	11.1

Section 2

Community Size	% M.D.'s (60 Ind. Pr.	% M.D.'s (60 Gr. Pr.
1500-6000	71.7	93.5
6000-10,000	62.9	100.0
10,000-20,000	71.2	93.1
20,000-38,000	79.0	89.3
Average	73.3	93.4

Section 3

Community Size	Net Change Ind. Pr.	Net Change Gr. Pr.	% of Est. Loss Offset* by Gr. Pr.
1500-6000	—44	+13	22.7
6000-10,000	—23	+10	46.2
10,000-2,000	—54	+22	33.9
20,000-38,000	+21	+15	53.5

*See footnote in Table VII—Section C.

TABLE VIII

Martin (3), discussing the 1959 AMA survey on group practice, pointed out that Oklahoma M.D.'s comprised 1.03% of all M.D.'s in the United States, and that 4.27% of all group practices existed in Oklahoma, including 2.45% of all M.D.'s in group practice in the U. S. and 12.0% of M.D.'s in Oklahoma. Although limited, the phenomenon of group practice is sufficiently developed in Oklahoma to lend itself to quantitative analysis in relation to the migration and distribution of physicians.

Table VII—Section A, presents the data on communities by increasing size, with hospitals, in which there are no organized groups practicing. The data is tabulated in a manner similar to that of Table VI in Part III of this series, with the exception of Column 4 which presents, in addition, the per cent replacement of M.D.'s lost between 1950 and 1959. The community size grouping differs also, being determined by the distribution of group practice according to community size. Although the data are not exactly comparable because of the latter qualification, it can be seen that the trends and sense are similar to those of the "urbanized" category in Table VI.

Table VII—Section B, presents the data on these communities with organized practicing groups arranged according to community size. The first three categories (1500 to 6000, 6000 to 10,000, 10,000 to 20,000 [1]) include communities with more than 40% of M.D.'s practicing in groups as of January 1, 1959. The last two categories (10,000 to 20,000 [2], and 20,000 to 38,000) include communities with less than 40% of M.D.'s in group practice in 1959. The former categories show a decreasing average per cent of M.D.'s in group practice with increasing community size. (Five communities were excluded, two in the 1500-6000 class because the per cent of physicians in group practice fell below 40% ; and three in the 10,000 to 20,000 [1] class because the data for group practice could not be clearly defined or verified.)

Comparison of the figures in Section B with those for the corresponding community size without group practice in Section A reveals:

(1) there are no obvious differences in the data for communities of 20,000 to 38,000 population with and without group practice.

(2) communities of 10,000 to 20,000 (2) population with an average of 26.1% of M.D.'s in group practice and those of

1500 to 6000 population with an average of 78.1% of M.D.'s in group practice show a greater per cent replacement and lower net loss of physicians than do their counterparts without group practice.

(3) the communities of the first three size categories, having the greater proportion of their M.D.'s in group practice, show a progressive net loss and decreasing per cent replacement in relation to increasing community size.

Table VIII presents a more detailed analysis of physician migration and replacement in those communities with group practice. The actual number of practicing M.D.'s is presented since the heavy weighting towards individual practice is obscured when comparison is made on a percentage basis.

It is immediately evident in Section 1 that what might be construed as a remarkable growth of group practice on a percentage basis (Col. 3 & 5) results from (1) the small number of physicians in group practice to those in individual practice, and (2) a concomitant decrease of physicians in individual practice and a smaller increase of those in group practice (Col. 2 & 4; Col. 3 & 5; and Section 3). Some might also contend that group practice represents an increased competitive hazard to individual practice. Be that as it may, it is obvious from our data that the decreasing proportion of M.D.'s in group practice, accompanying increasing community size (up to 20,000 population), Table VII—Section B, does not offset the losses from individual practice (Table VII—Section C). Furthermore, a chronological study indicates that the initiation or growth of group practice *follows* losses from individual practice.*

The lower average age of physicians in group practice, represented by the data in Table VIII—Section 2 probably reflects the relative newness of the phenomenon. This interpretation is strengthened by the fact that 10 of the 27 groups existing in these communities as of January 1, 1959 did not exist in 1950.

The phenomenon of group practice as it has developed and now exists in the state of Oklahoma suggests that the application of the large "clinic" principles of organization and the division of labor in the development of small groups is a means whereby medical practice may be carried on and stabilized in certain situations, which in themselves are uncongenial to individual practice. It thereby can continue, in Oklahoma at least, to provide an adaptive mechanism to meet public need for medical services in circumstances to which it is peculiarly fitted and to an extent appropriate to these needs.

The next and concluding article of the series will present data relevant to the relationship between hospital bed and physician distribution.

*Unpublished data on turnover of physicians, 1950 to 1959, in communities with practicing groups in 1959.

PRESIDENT'S LETTER



On May 13 and 14 I attended a conference on Prepaid Medical Care in Chicago, conducted by the American Medical Association. At this meeting were representatives of labor, the Blue Plans, doctors, commercial insurance companies and governmental agencies. It was most impressive to visit with over two hundred men from all parts of the country and listen to the panel discussions which the A.M.A. had set up. The discussions were lively and the exchange of information should be of great help to all areas.

The question of service versus indemnity plans cannot be settled by a conference or two of this sort, but the healthy discussions engendered do present both sides fairly. Indeed, it seemed to me that it was pretty definite that what works in one area will by no guarantee work successfully in another.

But aside from all the preferences for one type plan or another it has become pretty obvious that the third party is definitely here with the doctor and his patient and apparently is going to remain, even if it is only on a check list basis for proper processing of forms which the doctor has filled out. Insurance forms and itemized statements are the order of the day. In adjusting ourselves to this new phase of medical economics we must not become too disturbed over purely personal dislikes of procedures. The mechanics can be smoothed over in due process of time. Our concern is that we maintain our concepts of the ethical practice of medicine.

Walter E. Brown, M.D.

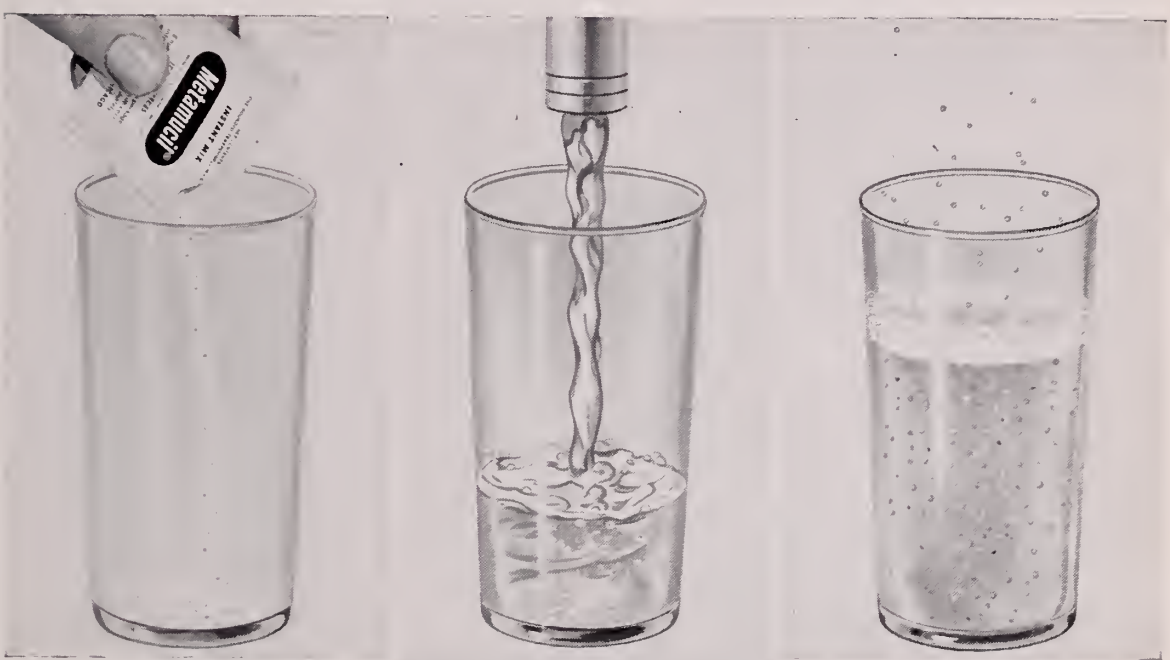
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News

Federal Employees' Health Plan To Begin July 1, 1960

Uncle Sam's gigantic health insurance plan for federal employees is scheduled to start on July 1, 1960. Proclaimed as the largest and most complex employer-sponsored health benefit program in the world, the Civil Service Commission's program will cover approximately 1,800,000 federal employees and their 2,200,000 dependents. Nationwide enrollment is being conducted during the month of June.

Prior to congressional approval of the Federal Employee Health Benefit Act of 1959, the government had not contributed to any employee health program. The act established a contributory plan similar to those used in many private industries. Estimates of the total employer-employee annual cost run around 250-million dollars, with the government putting up about 100-million dollars as its share.

Four Plans Available

Under the law, government workers may choose from four types of coverage: Service benefit plan, indemnity benefit plan, certain employee organization plans, and comprehensive medical plans of the group or individual-practice prepayment type. Nearly forty plans of various types have been approved to date, but most of them are small ones, primarily variations of existing employee organization plans to permit government contributions.

Service versus Indemnity

Two major plans which have been agreed upon are a Blue Cross-Blue Shield service program and a commercial indemnity plan, contracted by the Aetna Life Insurance Company. Aetna is expected to cooperate nationally with some two hundred private carriers who have expressed an interest in the underwriting. The Blues will farm their national contract out to the state plans, with local adjustments being made to conform to local policies.

Government participation in all types of plans will be uniform. Single employees will be entitled to a government contribution of $2.82 per month, married employees will receive $6.76 per month toward the cost of the plan they choose for their families. A female employee with a nondependent husband will receive $3.94 per month in federal assistance for a family coverage plan of her choice.

In both of the major plans, high and low benefit options are offered, but government contributions remain the same. Thus, an employee will first select between the service or indemnity options, then between the high and low benefit options. Through a payroll deduction system, the premium for the plan selected, less the government contribution, will be deducted from the employee's check on a weekly, bi-monthly, or monthly basis.

Total monthly premiums for the Aetna plan, before government contribution, are $5.64 (individual) and $13.52 (family) for the low benefit plan; $6.76 (individual) and $17.46 (family) for the high benefit plan.

Total monthly premiums for the Blue Cross-Blue Shield plan, before government contribution, are $5.64 (individual) and $14.21 (family) for the low benefit plan; $7.39 (individual) and $19.37 (family) for the high benefit plan.

The two major plans are both more liberal than the majority of existing plans (for example, there will be no waiting period for maternity benefits), but they are otherwise similar in benefits offered and premium costs to existing service and indemnity plans.

As Applied to Oklahoma

Oklahoma's 45,000 federal employees will basically have three major options to choose from in selecting a plan for themselves and their dependents: Blue Cross-Blue Shield service contract; Blue Cross-Blue Shield indemnity plan; and, the Aetna indemnity plan. Although some may participate in employee organization plans, such as various postal employees' plans, the majority will take either the Blue Cross or Aetna route. Some features of the major plans follow.

The Blue Cross Plan

While the Blue Cross-Blue Shield option is referred to nationally as a service plan, adjustments are being made in Oklahoma in order to conform to local Blue Shield policies. In the six Oklahoma counties where Blue Shield service contracts exist(Garfield, Grant, Rogers, Craig, Ottawa and Delaware) the high level benefit plan for Blue Shield will be offered to federal employees on a service basis. The low level benefit plan for Blue Shield will be offered on an indemnity basis, as it will be in the remaining seventy-one counties of the state where no service contract is in force. Federal employees in the "indemnity" counties may also purchase the high level benefit plan, but on an indemnity basis.

Thus, federal workers in the six counties may select from either service or indemnity as pertains to Blue Shield payments. Workers in the other counties may select either high or low benefit indemnity plans. Blue Cross will be on a service basis in all counties.

Premiums for the six service counties are as follows: Blue Cross-Blue Shield (service) $7.39 per month for an individual, and $19.37 per month for a family; Blue Cross-Blue Shield (indemnity) $5.64 per month for an individual, and $14.21 per month for a family.

For the remaining seventy-one counties, the rates for indemnity coverage are: Blue Cross-Blue Shield low benefit level plan—$5.64 per month for an individual, and $14.21 for a family; Blue Cross-Blue Shield high benefit level plan—$7.39 per month for an individual, and $19.37 for a family.

Benefits

In Oklahoma, there will be no income limits applicable to the service contracts in the six service counties. Thus, all employees who purchase the high level service plan will receive up to 120 days per admission in the hospital, other hospital supplies and services, and medical and surgical care without additional cost.

From an indemnity standpoint, the high option also covers 120 days per admission, but the low option is reduced to 30 days, as well as a lower fee schedule for physicians' services.

As previously indicated, hospital benefits will be on a service benefit basis for all Blue Cross plans, regardless of the county of residence. The "indemnity" plans, however, regardless of high or low level benefits, will enable the physician to bill the patient above the Blue Shield payment, if necessary. A partial list of the high and low option fee schedules is printed below (high option benefits will be accepted as full payment in service areas):

Sample Fee Schedule

SURGICAL BENEFITS	Actual charges up to HIGH OPTION	LOW OPTION
Appendectomy	$150.00	$100.00
Cholecystectomy	225.00	150.00
Breast Tumor removal	50.00	35.00
Breast, radical removal, both	300.00	200.00
radical removal, one	200.00	135.00
Submucous Resection	100.00	65.00
Tonsillectomy	50.00	30.00
Hemorrhoidectomy,		
internal and external	100.00	65.00
external only	40.00	25.00

Herniotomy, single, inguinal	150.00	100.00
Prostatectomy,		
transurethral resection	225.00	150.00
Thyroidectomy	225.00	150.00
Cataract, removal of,		
unilateral	225.00	150.00
Cystoscopy with biopsy	30.00	20.00
with stone removal	75.00	50.00
Brain cyst, excision of	300.00	200.00
Fracture of thigh (neck of		
femur), open reduction	300.00	200.00
Fracture of leg (tibia),		
closed reduction, simple	100.00	65.00
open reduction	150.00	100.00
Dilation and curettage	35.00	25.00
Circumcision (under 1 yr.)	10.00	5.00
Mastoidectomy, simple,		
both sides	225.00	150.00
Radical, one or both	300.00	200.00

For maternity benefits, the high option Blue Shield payments will be accepted as payment in full in the service counties. Under the high option, the physician will receive $90.00 for a normal delivery, versus $60.00 under the low option. Caesarean section fees are $150.00 and $100.00, high and low options, respectively. Miscarriage payments are $60.00 and $40.00.

The high and low option anesthesia fees will amount to 20% of the fee paid for either surgical or maternity care. A minimum payment of $15 is established under the high option, and $10 under the low option.

Hospital benefits under the high option plan pay up to $100. The low option pays $10 per day up to ten days.

The high option allowances for in-hospital medical care are: $15.00 first day; $10 second day; $4.00 third through tenth days; and, $3.00 thereafter. The low option allows $3.00 per day straight through.

The Aetna Plan

Under the Aetna, or national indemnity plan, the high level option pays the first $1,000.00 of calendar year's hospital room and board per person, and 80% of any charge over that amount. For other hospital expenses, as well as medical expenses, including operations, nursing care, and physicians' visits, the high option pays 80% of charges over the first $50.00 per person.

The low option pays the first $250.00 of the calendar year's hospital room and board per person, and 75% of any charge over that amount. For other hospital and medical expenses, the low option pays 75% of charges over the first $50.00 per person.

For maternity benefits, the high option pays $15.00 a day for 10 days for hospital expenses. The obstetrician receives up to $90.00 for normal delivery, $150.00 for caesarean, and $60.00 for miscarriage. Employees pay the rest. The anesthetist is paid up to $18.00 for normal delivery, $30.00 for caesarean, and $12.00 for miscarriage.

The low option maternity benefits include up to $10.00 a day for 10 day's hospital care, $60.00 for normal delivery, $100.00 for caesarean, and $40.00 for miscarriage. Anesthesia fees are $12.00 for normal delivery, $20.00 for caesarean, and $8.00 for miscarriage.

Major Medical

Both Blue Cross-Blue Shield and Aetna offer supplemental, or maximum benefits. Aetna allows $30,000 (high option) and $10,000 (low option) for each person, but pays only 75% or 80% of charges incurred (low and high option). The Blues offer high and low maximums of $10,000 and $2,500 during any one benefit period, with the benefit period ending twelve months after hospital admission, or after ninety days of non-coverage following discharge. The maximums for two or more benefit periods are $20,000 (high) and $5,000 (low) for each person. A deductible is applied before supplemental benefits begin. It is $100 for the high option plan, and $200 for the low option plan.

Determination of Eligibility

Information regarding the eligibility of employees and their families will be sent to physicians and hospitals from the two principal carriers.

Councils, Committees Announced for 1960-61

President Walter E. Brown, M.D., has released information regarding Council and Committee appointments for the term of his administration.

The organizational structure is essentially the same as employed last year by Alfred T. Baker, M.D., immediate past president. Members of the OSMA will recall that Doctor Baker initiated a committee reorganization plan which grouped all association committees under an overlay of five principal "councils." The purpose of the experimental plan was twofold: To conform as closely as possible with the organization of the American Medical Association; and, to initiate much-needed streamlining and efficiency in handling the myriad projects and problems now confronting organized medicine at the state level.

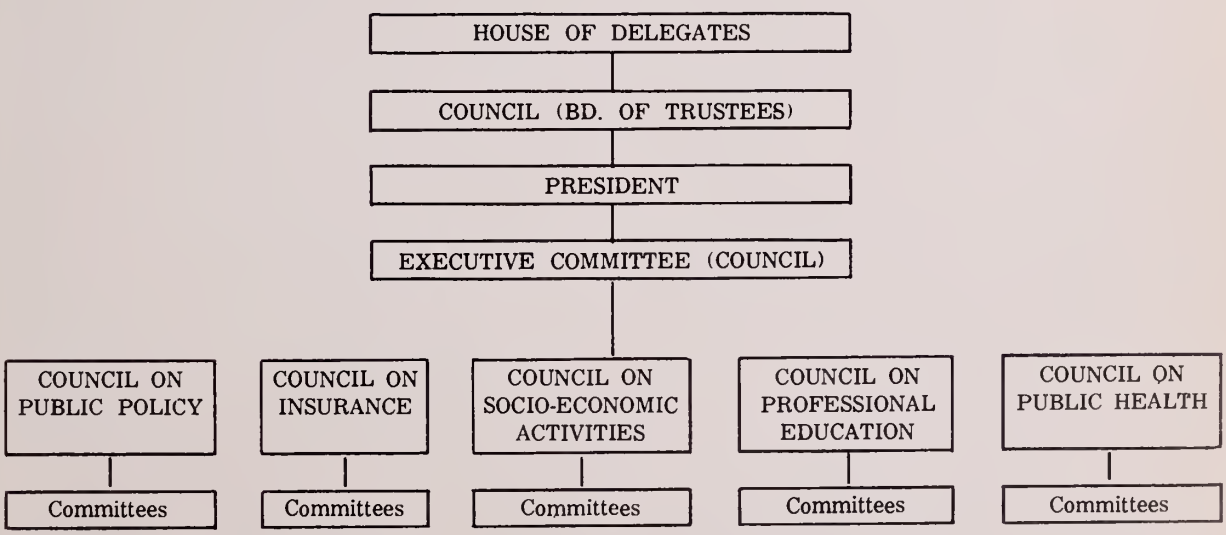
How It Works

In contrast to the previous system of simply using special committees under the direction of the President and policy-making bodies, the council system enables the President to delegate major activities to the wisdom and experience of the five principal councils. The councils, in turn, will serve in a supervisory capacity for all committees under their direction, and also serve as a liaison link between the committees and the President and policy-making bodies. The plan is designed to relieve the President from being singularly responsible for the coordination and consistency of some twenty to forty committees which are appointed annually.

Trial Plan

Started by Doctor Baker as a one year trial plan, to be formalized if proven successful, the plan is now being tested for an additional year before final action. If finally adopted by the policy-making bodies, the formal creation of the five councils will necessitate re-naming the association Council to the Board of Trustees. This action would obviously eliminate organizational confusion. It would also perhaps be necessary to re-designate the Executive Committee as the Executive Council.

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Mr. Tom Brett, Oklahoma City, Director of Civil Defense of Oklahoma
Henry D. Dericks, Commander, United States Navy, Tulsa
Kirk T. Mosely, M.D., Commissioner of Health, Oklahoma City

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of the

OKLAHOMA STATE MEDICAL ASSOCIATION

1960-61

PRESIDENT—Walter E. Brown, M.D., Tulsa

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DELEGATE TO THE A.M.A.—Wilkie D. Hoover, M.D., Tulsa

ALTERNATE DELEGATE TO THE A.M.A.—Joe L. Duer, M.D., Woodward

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Sentinel

'Care for Aged' Bills Falter

The hue and cry for a Federal health care plan for the aged appears to be waning in the closing weeks of Congress—or, at least the current activities in this area are taking more conservative trends.

Whether the slackening of interest is a result of global concern caused by the U-2 incident, or whether it is simply a realization on the part of Congressmen that the old folks themselves are not pushing for the legislation, is a matter of speculation. In regard to the latter development, reliable reports indicate that pro-legislation mail being received in Washington contains few letters from the elderly, and legislators are beginning to take note that the true source of the agitation is the AF of L-CIO. Moreover, even the retired union workers are not responding to labor leaders' demands to bombard their lawmakers.

● BULLETIN ●

On June 9, Delegates to the Oklahoma White House Conference on Aging rejected Social Security-type legislation; endorsed voluntary programs.

On the other side of the fence, rank and file members of professional associations, farm organizations and business and civic groups are flooding their respective Congressmen with sensible arguments of opposition to the social welfare movement. Thus, the wooing of the aged voting bloc has lost some of its gloss, and the outlook today is much more favorable than it was a few months ago when Democratic candidates for the presidency pledged allegiance to the Forand movement by proposing various and sundry bills of their own.

Current Status

Although the de-emphasis of interest is encouraging, the matter is far from being settled. The present picture is a complex one, clouded with the introduction of many bills.

The Ways and Means Committee, led by Representative Wilbur Mills (D. Ark.), has rejected all proposals submitted to it, including the Forand Bill and the Administration's plan. In the latter regard, the Administration recently suggested a Federal-state partnership to pay for co-insurance, deductible-type health and hospital coverage to protect retired persons against catastrophic medical expenses. The program, which would cost state and Federal governments $1,200,000,000 annually, would require the "insureds" to pay $24 each per year toward the cost. They would receive 80% of the cost of medical, dental, hospital, nursing home and other services, after paying a $250 deductible for individuals and a $400 deductible for families. Some observers feel that the administration submitted this plan as a political coup, knowing it would fail to receive the support of anyone.

Looming as the biggest threat of the moment is the combine of Speaker Sam Rayburn and his choice for President, fellow Texan Senator Lyndon B. Johnson, who is also the Democratic majority leader. For months, both opposed the use of the Social Security system as a vehicle for financing a health care plan, but they have recently made an abrupt shift.

Influenced by the political storm and the need for a strong election issue, they are now throwing their considerable weight in support of legislative action. Ways and Means Committee Chairman Mills is doing his best to hold the line, but there is always the danger that he will weaken under the pressure of Rayburn.

Watered-Down Compromise

With all of the push and pull and general uncertainty, the most likely legislation at the moment would be the Ways and Means authored bill to extend the Old Age Assistance medical care program to include the aged who fall on the borderline of welfare eligibility. While the American Medical Association is not actively soliciting such a program, it has let it be known that the principle involved does not appear to be contrary to official association policy.

Proceedings of the 54th Annual Session of the House of Delegates of the Oklahoma State Medical Association

Opening Session*

The 54th Annual Session of the House of Delegates of the Oklahoma State Medical Association was called to order Sunday, May 1, 1960, at 10:00 a.m., in the Mirror Room of the Municipal Auditorium, in Oklahoma City, Oklahoma, by Clinton Gallaher, M.D., Shawnee, Speaker of the House of Delegates.

Doctor Charles E. Green, Lawton, gave the invocation.

Doctor Gallaher announced that all voting Delegates should be seated in the front section of the room, where tables are provided, and all others should please find chairs toward the back of the room.

Doctor Gallaher asked the Credentials Committee if a quorum were present. The Credentials Committee announced a quorum present.

The Speaker announced the appointment of the following working committees of the House of Delegates:

Credentials Committee

C. Riley Strong, M.D., El Reno (Chairman)
Hugh Perry, M.D., Tulsa
Peter Russo, M.D., Oklahoma City

Resolutions Committee

E. C. Mohler, M.D., Ponca City (Chairman)
C. M. Hodgson, M.D., Kingfisher
Joe L. Duer, M.D., Woodward
Wilkie D. Hoover, M.D., Tulsa
Ross Deputy, M.D., Clinton

Sergeants at Arms

Paul Kernek, M.D., Holdenville
Francis R. First, M.D., Checotah

Tellers

Thomas E. Rhea, M.D., Idabel
Virgil Ray Forester, M.D., Oklahoma City
Ollie McBride, M.D., Ada

Constitution and By-Laws

William T. Gill, M.D., Ada (Chairman)
Marshall O. Hart, M.D., Tulsa
A. C. Lisle, M.D., Oklahoma City

Parliamentarian

William T. Gill, M.D., Ada

Doctor Gallaher, as the next order of business, began the introduction of guests.

Doctor Gallaher introduced Mrs. Clifford M. Bassett of Cushing, President of the Woman's Auxiliary to the Oklahoma State Medical Association.

*Proceedings of the closing session will appear in the July *Journal*.

Mrs. Bassett addressed the House of Delegates and informed them of the activities of the Auxiliary during the past year. One point she stressed was the tremendous increase in membership of the Auxiliary in the last few years.

Mrs. Bassett introduced Mrs. Virgil Ray Forester the new President of the Woman's Auxiliary.

Doctor Gallaher next introduced Melvyn Brill, President of the Oklahoma Chapter of the Student American Medical Association, who brought greetings to the House of Delegates members from his fellow members. He introduced Rex Baggett, his successor.

Doctor Gallaher then introduced Grady F. Mathews, M.D., Commissioner of Health of the Oklahoma State Health Department. The House of Delegates members gave Doctor Mathews a rousing ovation for his twenty years of service as Commissioner.

Doctor Gallaher asked the pleasure of the House with regard to the reading of the Minutes of the last Annual Session, which were published in the Oklahoma State Medical Association Journal.

It was moved that the minutes be adopted as published, and that they dispense with their reading at this session. The motion was duly seconded and carried.

Doctor Gallaher announced that the next order of business would be the nomination of officers. He advised that Councilor Districts Numbers 2, 5, 8, 11, and 14 would elect councilors and District Number 12 would elect a councilor to fill the unexpired term of Doctor William T. Gill of Ada who had resigned. Doctor Gallaher read the counties included in these districts and presented the names of the incumbents. The Speaker again announced that the Constitution and By-Laws adopted at the 1958 meeting changed the number of consecutive terms a Councilor could serve from two (2) to three (3) consecutive terms, and that all the incumbents were eligible for re-election.

Doctor Gallaher announced that the House would recess for ten minutes to allow the councilor districts to caucus.

Following the brief recess, the House reconvened and Doctor Gallaher asked for any additional announcements or introduction of guests.

There being none, Doctor Gallaher then read a telegram from Louis H. Bauer, M.D., Secretary General of the World Medical Association, extending greetings and best wishes for a successful meeting.

The Speaker then declared the House of Delegates open for nomination for the office of President-Elect,

(Continued on Page 471)



Fifteen Attend Past Presidents' Breakfast

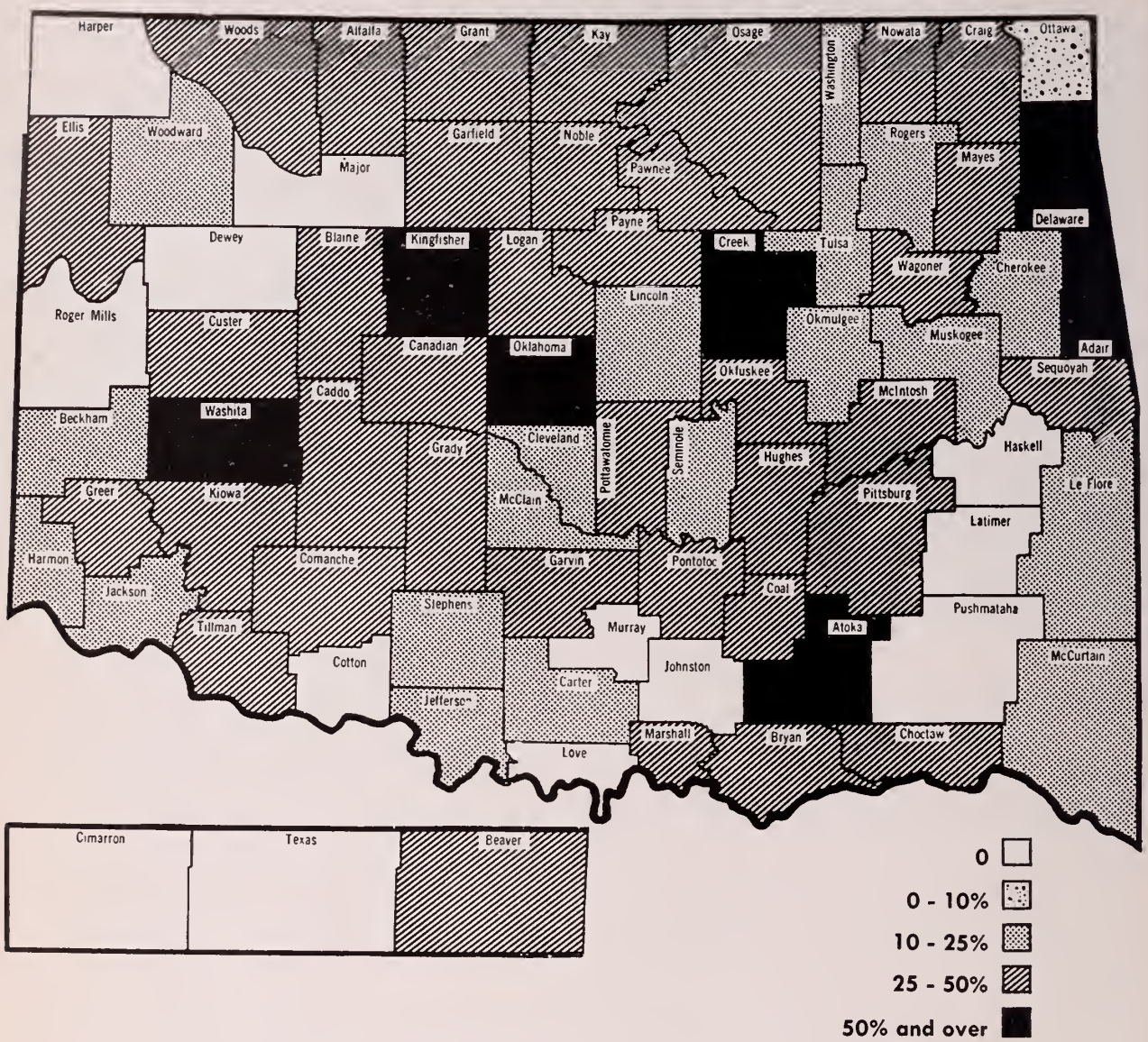
Past Presidents, fifteen strong, showed up in a new setting for the traditional breakfast during the state meeting. The Oklahoma Medical Research Foundation, an organization planned and supported by the medical profession, was host for the breakfast. Pictured above (top row), are John Flack Burton, M.D., Oklahoma City; R. Q. Goodwin, M.D., Oklahoma City; Henry K. Speed, M.D., Sayre; Louis H. Ritzhaupt, M.D., Guthrie; E. C. Mohler, M.D., Ponca City; Paul B. Champlin, M.D., Enid; (bottom row) Bruce R. Hinson, M.D., Enid; C. E. Northcutt, M.D., Ponca City; T. H. McCarley, M.D., McAlester; George H. Garrison, M.D., Oklahoma City; E. S. Lain, M.D., Oklahoma City; and P. P. Nesbitt, M.D., Tulsa. Also attending but not pictured, were Henry H. Turner, M.D., Oklahoma City; Chester McHenry, M.D., Oklahoma City; and Walter E. Brown, M.D., Tulsa. In addition, Mr. John Kirkpatrick acted as official host and R. Palmer Howard, M.D., represented the scientific staff of the Foundation. Kirkpatrick is a member of the executive committee of the Foundation. (John E. McDonald, M.D., Tulsa, came the following morning, but was not served.)

Appointments for Medical Officers In Reserve Components of the Army

The 45th Infantry Division, Oklahoma National Guard, announced that there are now some vacancies in the Guard for appointments to the Medical Corps. The appointments are from the rank of Lieutenant through Lt. Colonel depending on education, medical or military experience. This is an excellent opportunity for recent graduates to fulfill their military obligation and continue their hospital training or private practice. This also is an opportunity for the busy practitioner who desires a change of scenery and respite from the ringing telephone for two weeks each August at the summer training camp.

In addition to the above advantages, an officer earns retirement credits which will provide a very good income at age 60.

For further details contact Doctor (Colonel) Claude B. Knight, Wewoka, Oklahoma, Division Surgeon, or Doctor (Colonel) John B. Miles, 702 W. Central, Anadarko, Oklahoma.



54th ANNUAL MEETING ATTENDANCE, BY COUNTY

Each year, annual meeting officials are presented with the ever-increasing challenge of developing and promoting a scientific and social program which will attract the interest of OSMA members. The yardstick for this year's successes and failures is graphically depicted above on a county basis.

It appears significant that, with the exception of the host society, geographic relationship to the annual meeting site has little effect upon attendance percentages from the county medical societies. Neither can any conclusions be drawn from the standpoint of county society size.

The seven societies showing over 50% registration run the gamut in both geographic location and size. Although a large attendance would be normally expected from the host society, the fact that Oklahoma County supplied 310 registrants, almost half of a total of 675, is a surprising revelation.

In examining the next ten largest societies, representing an aggregate membership of 723, five of them showed less than 25% registration, and five less than 50%. If 50% participation could have been achieved in these ten counties alone, the 54th Annual Meeting registration would have reached 860—50% of state association membership and a modern-day record.

House Staff Physicians Win Honors

Honors for "best paper of meeting" at the sixth annual Oklahoma Association of House Staff Physicians program were shared by H. Earl Ginn, M.D., resident in medicine, and William E. Price, M.D., resident in surgery, both of the University of Oklahoma Medical Center.

The Oklahoma City Chapter of the Medical Service Society of America, which established the award last year, will give each physician two plaques, one for his hospital and one for himself.

Doctor Ginn presented a paper on "An Investigation of Renal Tubular Defects Associated with Magnesium Depletion in Rats." Doctor Price's award-winning report was on the mechanism of antral regulation of gastric secretion. Their papers also were judged the best in surgery and in medicine.

T. E. Acers, M.D., of the University of Oklahoma Hospitals, was given the Oklahoma Academy of General Practice citation for the best paper by an intern. Subject: Experimental Supravalvular Mitral Stenosis in the Dog.

The Oklahoma chapter of the American College of Surgeons again selected six outstanding papers and will invite the authors to present them before the College this autumn. Winners were: Doctor Price; Charles A. Rockwood Jr., M.D., orthopedic surgery resident (on non-tuberculosis psoas abscess); Clark A. Grimm, M.D., radiology resident (endometrial carcinoma), Frank Howard, M.D., surgery resident (complications of surgical wounds), William J. Waskowitz, M.D., orthopedic surgery (malignant soft tissue tumors), all of the OU Medical Center, and Joseph W. Stafford, M.D., surgery resident, St. Anthony Hospital, Oklahoma City (carcinoma of the gall bladder—fact or fancy?).

Richard Bottomley, M.D., resident in medicine at the Medical Center, won the Medical Service Society award for the best basic science presentation. He reported findings on the effect of cortisone on the in-vitro incorporation of C-14-formate into the protein and nucleic acid fractions of leukemic lymphocytes.

The Oklahoma City Pediatric Society citation went to Mary I. Abbott, M.D., Medical Center pediatric resident, for a paper entitled: "Measles (Rubeola) Vaccine: An Evaluation of Reaction Rates and Antibody Response to the 3 Types of Vaccine."

Doctor Grimm's paper on a 13-year review of endometrial carcinoma also took the Oklahoma State Radiological Society prize.

Guest speakers at the May 6 meeting in the Veterans Administration Hospital were Isadore Dyer, M.D., head of the Department of Obstetrics and Gynecology, Tulane University School of Medicine, and J. Willis Hurst, M.D., head of the Department of Internal Medicine, Emory School of Medicine.

Doctor Howard, the 1960 program chairman, was elected Association president, succeeding Doctor Ginn.

Next year program chores will be divided among Howard Keith, M.D., and William B. Shelton, M.D., both residents at OU Medical Center, and Frank Clingan, M.D., resident, Hillcrest Medical Center, Tulsa. Doctor Rockwood was named social chairman, and Doctor Bottomley and his wife, Sylvia S. Bottomley, M.D., also Medical Center residents, will handle the secretary-treasurer job.

Pittsburg County M.D.'s Help Tornado Victims

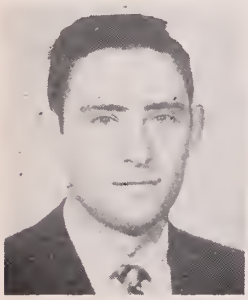
An outstanding public relations gesture on the part of the Pittsburg County Medical Society was recently revealed.

C. K. Holland, M.D., President of the society, reported that physicians' fees from prepaid insurance coverage for medical care rendered to Wilburton tornado victims would be earmarked for the creation of a fund to purchase emergency electrical power equipment for the McAlester hospitals. Patients without health insurance will not be billed for services rendered to them.

All funds accruing from this endeavor will be used to modernize and upgrade the present emergency electrical facilities, Doctor Holland said. The medical society pointed out to the community the extreme importance of the hospitals having adequate emergency power to operate such services as x-ray, laboratory, operating rooms, elevators and refrigeration equipment.

Brill Named to SAMA Post

The Oklahoma SAMA chapter again has won representation on the national Student AMA executive council. The OU delegation bagged two more offices at the Los Angeles convention, presided over by Oklahoma student William R. Kirkham (M.D., 1960).



BRILL

Melvyn Brill, now a senior, was named Region 6 vice president. Brill, of Lawton, is outgoing president of the OU chapter. New chapter president is Rex Baggett, Oklahoma City, now a junior. Mrs. Baggett was elected second vice president of the national Auxiliary.

Doctor Kirkham served as national president the past year and Mrs. W. Stanley Muenzler, wife of a graduating senior, was national Auxiliary chief.

The delegation to the April sessions also included Baggett, Ronald White, chapter vice president, Sam Hamra, secretary-treasurer, Jerry Blankenship, James Garner, Mrs. Blankenship and Mrs. Kirkham.

New FAA Rule Affects Physicians

Effective June 15, 1960, the Federal Aviation Agency will require that student and private pilots be given their medical examinations by designated medical examiners. This rule reinstates a practice which was in effect from 1926 until 1945.

In announcing the reestablishment of this practice, Doctor James L. Goddard, the Civil Air Surgeon, has emphasized his previous statements that any physician may be considered eligible for designation as an examiner.

His statement, made public February 11, 1960, follows:

"In order to have a better understanding of the proposed rule, I wish to point out that it is designed to accomplish the following needed improvements in the administration of the Agency's medical certification program.

1. To maintain a group of medical examiners who are clearly responsive to the needs of public safety in the performance of examinations and the issuance of medical certificates to airmen.

2. To permit the administration of training programs to maintain the quality of performance of medical examiners and to permit the dissemination of special instructions pertaining to the needs of civil aviation.

3. To bring into the program those physicians who have the professional qualifications and a demonstrated interest in the medical certification field.

4. This would permit the designation of any qualified physician who, by his application, has demonstrated interest in the program."

Those physicians in localities where flying activities are conducted may wish to consider filing an application for designation by writing to the Civil Air Surgeon, Federal Aviation Agency, Washington 25, D. C.

Designation as an aviation medical examiner will qualify the designee to examine both Class II (commercial) and Class III (student and private) airmen, including control tower operators. Instructions concerning the required procedures, standards, and equipment will be supplied to those who apply.

Since commercial and airline transport pilots have always been required to obtain examinations from specifically selected physicians, there are presently some 2,000 aviation medical examiners previously designated and located throughout the country. Expanding aviation activities will result in a continuing need for additional examiners. There are at present some 400,000 active civil airmen of whom approximately 240,000 are examined each year.

Date Set for McAlester Symposium

The McAlester Clinic Foundation and the University of Oklahoma School of Medicine, Department of Post Graduate Education, will present the Sixth Annual Southeastern Oklahoma Clinical Symposium in McAlester July 30-31, 1960.

Guest speakers and their topics will be: R. S. Griffith, M.D., Department of Medicine, University of Indiana, "Increasing the Effectiveness of Antibiotics" and "Watch Out! You're Using An Antibiotic"; A. Ford Wolfe, M.D., Department of Medicine and Allergy, Scott-White Clinic, "Practical Points in Control of Asthma"; G. Rainey Williams, M.D., Department of Surgery, University of Oklahoma Medical School, "Advances in Treatment of Cardiac Arrest" and "Management of Chest Injuries"; Sylvia Richardson, M.D., Department of Pediatrics, University of Oklahoma Medical School, "Learning Difficulties and Central Nervous System Dysfunction"; Tom S. Gafford, M.D., Pathologist, Muskogee, Oklahoma, "Use and Abuse of Blood Transfusions" and Clinical Pathological Conference; William R. Richardson, M.D., Department of Pediatric Surgery, University of Oklahoma Medical School, "Management of Gastrointestinal Bleeding in Infants and Children" and "Surgical Emergencies in the Newborn"; George M. Brown, Jr., M.D., Department of Surgery, McAlester Clinic, "A Ten Year Appraisal of Routine Operative Cholangiography"; Samuel E. Dakil, M.D., Department of Otolaryngology, McAlester Clinic, "Recent Advances in Otolaryngology."

A special feature on the program will be a Panel on Marital Counseling, by four members of the Oklahoma City Family Clinic: Mr. Bliss Kelley, Attorney; N. F. Van der Barkett, M.D., Physician; Rev. Louis DeFreese, Minister, and Mrs. Scott Downey, Financial Advisor.

Entertainment for the ladies has been planned.

A complete program and reservations may be obtained by writing to Mr. Jewell M. Green, Jr., Business Manager, McAlester Clinic, McAlester, Oklahoma.

Five Oklahomans Certified By OB-GYN Board

A recent report from the American Board of Obstetrics and Gynecology announced that five Oklahoma physicians were certified by the Board on April 15, 1960. The physicians are: Paul Albert Bischoff, M.D., Tulsa; Charles R. Cochrane, M.D. (Major—Medical Corps), Ft. Sill; Joseph W. Funnell, M.D., Oklahoma City; Billie Gene Henley, M.D., Lawton; and, Edwin Patrick Shanks, M.D., Enid.

Certifications following examination are made once a year by the Board.

Doctor Williams Accepts Mental Health Post

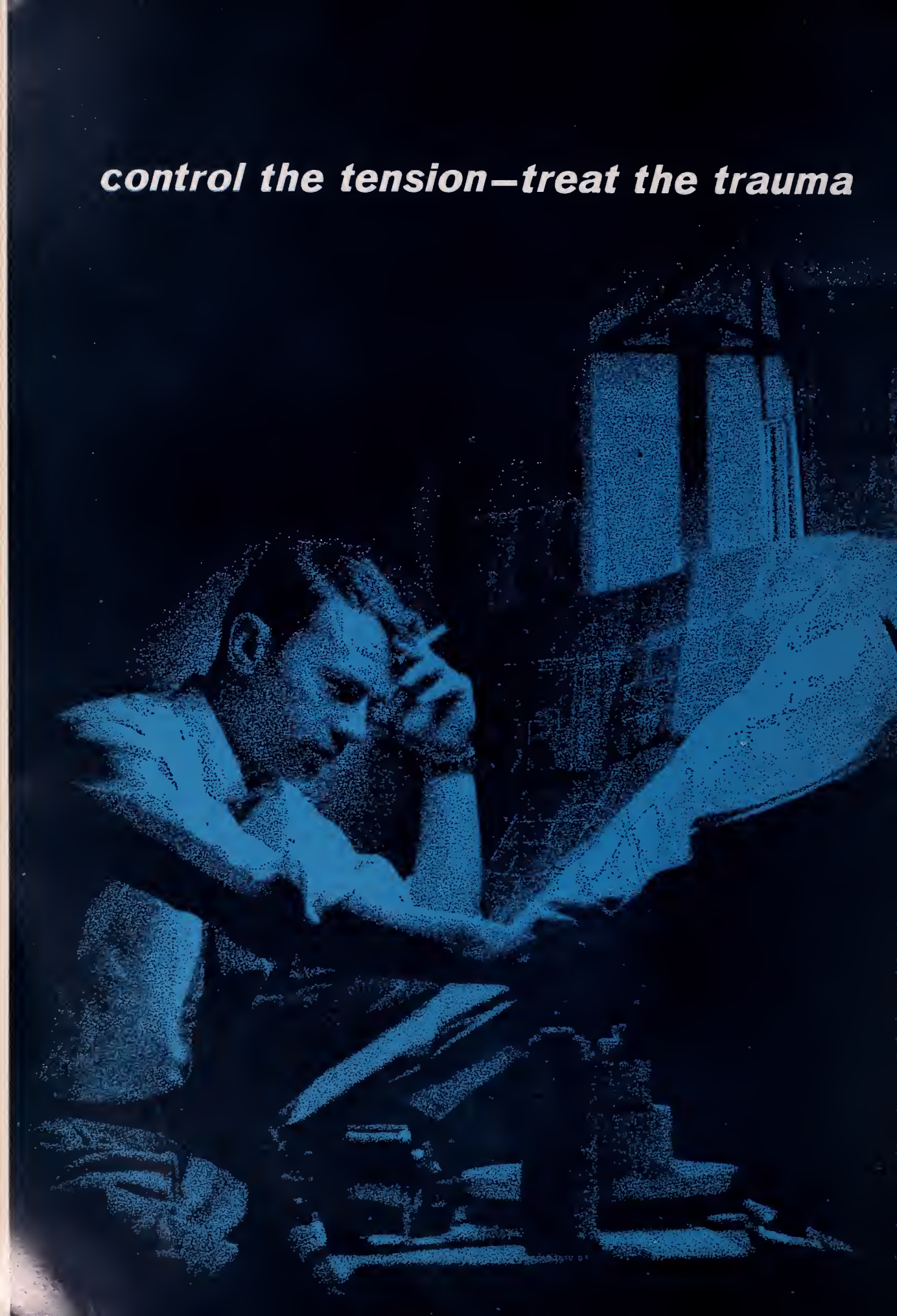
T. Glyne Williams, M.D., Assistant Professor of Psychiatry and Public Health at Yale University, will report for duty on July 1 as Oklahoma's new Commissioner of Mental Health. He is the long-sought replacement for Hayden Donahue, M.D., who resigned the position last fall for additional training and private practice. Since Doctor Donahue's departure, Rheba Huff Edwards, M.D., has been serving as Acting Commissioner.

Doctor Williams is not a neophyte in psychiatric administration. At the present time, he is Director of the Yale Pilot Study for Psychiatric Administration; Consultant to the Connecticut Department of Mental Health; Inspector (of mental hospitals), Central Board of Inspection, American Psychiatric Association; and, Professional Advisory Board Member, Connecticut Mental Health Association.

His professional qualifications include certification by the American Board of Psychiatry and Neurology and by the American Psychiatry Association as a Mental Health Administrator.

The 44-year-old physician received his B.S. degree from the University of the South, Sewanee, Tennessee in 1939, and his medical degree from Vanderbilt University School of Medicine, Nashville, Tennessee in 1943. In 1944-45, he was the psychiatrist for the U.S. Naval Hospital, Norman, Oklahoma.

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greater flexibility in the control of tension, hypermotility and excessive secretion in gastrointestinal dysfunctions

PATHIBAMATE combines two highly effective and well-tolerated therapeutic agents:

meprobamate (400 mg. or 200 mg.) widely accepted tranquilizer and...

PATHILON (25 mg.)—anticholinergic noted for its peripheral, atropine-like action, with few side effects.

The clinical advantages of PATHIBAMATE have been confirmed by nearly two years' experience in the treatment of duodenal ulcer; gastric ulcer; intestinal colic; spastic and irritable colon; ileitis; esophageal spasm; anxiety neurosis with gastrointestinal symptoms and gastric hypermotility.

Two dosage strengths—PATHIBAMATE-400 and PATHIBAMATE-200 facilitate individualization of treatment in respect to both the degree of tension and associated G.I. sequelae, as well as the response of different patients to the component drugs.

Supplied: **PATHIBAMATE-400**—Each tablet (yellow, 1/2-scored) contains meprobamate, 400 mg.; PATHILON tridihexethyl chloride, 25 mg.

PATHIBAMATE-200—Each tablet (yellow, coated) contains meprobamate, 200 mg.; PATHILON tridihexethyl chloride, 25 mg.

Administration and Dosage: **PATHIBAMATE-400**—1 tablet three times a day at mealtime and 2 tablets at bedtime.

PATHIBAMATE-200—1 or 2 tablets three times a day at mealtime and 2 tablets at bedtime.

Adjust to patient response.

Contraindications: glaucoma; pyloric obstruction, and obstruction of the urinary bladder neck.

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OB-GYN Certification

Applications for certification in the American Board of Obstetrics and Gynecology, new and reopened, Part I, and requests for re-examination in Part II are now being accepted. Deadline for receipt of applications is August 1, 1960. Application fee ($35.00), photographs, and lists of hospital admissions must accompany all applications.

As announced previously, after July 1, 1962, the board will require a minimum of three years of approved progressive residency training to fulfill the requirements for admission to examination. After that date, training by Preceptorship will no longer be acceptable.

Address inquiries to Robert L. Faulkner. M.D., 2105 Adelbert Road, Cleveland 6, Ohio.

Bus Drivers Will Need Physical Exams

Beginning with the school year July 1, 1960, all public school bus drivers will be required to have a medical examination and an opinion as to their competency, both physically and emotionally, to drive a school bus.

Forms for recording examination are being supplied to local school districts by the State Department of Education. During the summer months, bus drivers will be coming to physicians for this examination.

There are 3,300 school buses in operation in the State carrying from 20 to 75 children each. School bus transportation has been and still is one of the greatest factors in expanding educational opportunities by bringing the rural and suburban children to school. The resulting consolidation of schools, while providing finer physical facilities and an enriched curriculum, compels many pupils to ride long distances over roads and highways of ever increasing hazards. The educational system, therefore, has an obligation to provide every precaution to safeguard those pupils who are so transported. It is a recognized fact that the most important factor in accomplishing a greater degree of safety is a more competent school bus driver.

Health certificates signed by the physician will be an important part of the evaluation of competency of the men and women who will be driving buses transporting children to and from school. Interested officials urge physicians to lend their support to this worthwhile endeavor and to be thorough and objective in their evaluation of individuals presenting themselves for examination.

Have You Heard?

CARL WHINERY, M.D., a graduate of the University of Oklahoma School of Medicine, recently opened offices at 106 South Fourth Street in Sayre, Oklahoma. Doctor Whinery had practice in Yosemite, California since 1958.

ROBERT O. RYAN, M.D., Norman physician, was elected mayor of Norman by the City Commission on May 10. Doctor Ryan's election, his first as mayor, marked the midpoint of his third two-year term on the Commission.

DAVID D. FRIED, M.D., Bethany physician, left on May 12 for Ogbonosho, Nigeria, West Africa, where he will spend the next three months in medical missionary work. Accompanying Doctor Fried were his wife and three children.

KENNETH L. PEACHER, M.D., a former El Reno resident who has been practicing medicine in Waynoka for the past 11½ years, has returned to El Reno and will open his offices in the Professional Building.

NOEL MILLER, M.D., his wife, BONNIE MILLER, M.D., and DONALD D. COLLINS, M.D., will establish a clinic in Okemah in June. All three physicians are native Oklahomans and graduated from the University of Oklahoma School of Medicine. The new clinic will be known as the Collins-Miller Clinic.

Coming Meetings

ROCKY MOUNTAIN CANCER CONFERENCE

July 20, 21, 1960 — **Denver Hilton Hotel**
Denver, Colorado

The 14th Annual Rocky Mountain Cancer Conference will be held at the Denver Hilton Hotel in Denver, July 20, 21, 1960. Details may be obtained from the Rocky Mountain Cancer Conference, 835 Republic Building, Denver 2, Colorado.

DERMATOLOGY
for
GENERAL PRACTITIONERS

July 21-23, 1960 — **Denver, Colorado**

A postgraduate course in Dermatology for General Practitioners will be offered by the Office of Postgraduate Medical Education, University of Colorado Medical Center, July 21-23, 1960 in Denver. Details may be obtained by writing to the postgraduate office, 4200 East Ninth Avenue, Denver 20, Colorado.

INTERNAL OPHTHALMOLOGY
and
Summer Convention of the Colorado Ophthalmological Society

July 5-8, 1960 — **Aspen, Colorado**

The second annual postgraduate course in Ophthalmology and the summer convention of the Colorado Ophthalmological Society will be held July 5-8, 1960 in Aspen, Colorado. Detailed information is available from the Office of Postgraduate Medical Education, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver 20, Colorado.

POSTGRADUATE COURSE IN PEDIATRICS
The University of Colorado School of Medicine

September 1-6, 1960 — **The Stanley Hotel**
Estes Park, Colorado

The Department of Pediatrics and the Office of Postgraduate Medical Education, University of Colorado School of Medicine will present a Course in Pediatrics at the Stanley Hotel, Estes Park, Colorado, September 1-6, 1960.

SECOND ANNUAL
OREGON CANCER CONFERENCE

July 7-8, 1960 — **Portland, Oregon**

The second Annual Oregon Cancer Conference will be held in Portland, July 7-8, 1960. A copy of the complete program may be obtained by writing to Roscoe K. Miller, Executive Secretary, Oregon State Medical Society, 2164 S. W. Park Place, Portland 5, Oregon.

SOUTHERN TRUDEAU SOCIETY
and
SOUTHERN TUBERCULOSIS CONFERENCE

September 14, 15, 16, 1960 — **Hotel Francis Marion**
Charleston, South Carolina

The 1960 Annual Meeting of the Southern Trudeau Society and the Southern Tuberculosis Conference will be held on September 14, 15, and 16, 1960 at the Hotel Francis Marion, in Charleston, South Carolina. Information may be obtained from Judson M. Allred, Jr., Secretary-Treasurer, Southern Tuberculosis Conference, P.O. Box 9865, Northside Station, Jackson, Mississippi.

FOURTH NATIONAL CANCER CONFERENCE

September 13-15, 1960 — **Minneapolis, Minnesota**

The Fourth National Cancer Conference will be held September 13-15, 1960 in Minneapolis, Minnesota. Further information may be obtained by writing to Roald N. Grant, M.D., Coordinator, American Cancer Society, Inc., Medical Affairs Department, 521 West 57th Street, New York 19, New York.

6th INTERNATIONAL CONGRESS OF
INTERNAL MEDICINE

August 24-27, 1960 — **Basle, Switzerland**

The 6th International Congress of Internal Medicine will convene in Basle, Switzerland, August 24-27, 1960. Simultaneous translation for the principal speakers will be given in German, French and English. Programs and registration forms are available from the 6th International Congress of Internal Medicine, Steinentorstrasse 13, Basle 10, Switzerland.



Unless your practice is limited to bacteriology . . . or your patients are all in the upper income brackets . . . you have doubtless received complaints about the cost of the medication you prescribe.

what your patient

gives...and gets

Some of these complaints can probably be dismissed lightly as coming from cranks, who would complain about your fee for a midnight house call to save the life of a dying child. Others, however, are made seriously by thoughtful patients and deserve an answer in kind. You know what the patient gets from his pharmacist because you have prescribed it. Do you also know that the average cost of a prescription is about $3.00? Only about one in 100 costs $10.00 or more, and 3 out of 5 of the prescriptions are under $3.00. These figures are based on retail prices. They include the manufacturer's research, development, and manufacturing costs and all distribution costs of the wholesale and the retail druggist. Only you and your patients can judge whether today's drugs at these prices represent a fair *quid pro quo,* an equitable balance between what is given and what is received.

This message is brought to you by 138 producers of prescription drugs as a service to the medical profession and in the same spirit, it is carried by this publication. For additional information, please write Pharmaceutical Manufacturers Association, 1411 K Street, N.W., Washington 5, D.C.

Auxiliary News

Preserve and Enhance

"Preserve and enhance the heritage of American Medicine," the theme for 1960-61, was presented by Mrs. William Mackersie, President-Elect of the Woman's Auxiliary to the American Medical Association, during the opening session of our state convention.

The theme is from National; however, our state interest lies in its development and interpretation at the county level. Next spring at state convention we will have again the opportunity of hearing with pride the accomplishments of county auxiliaries, both large and small.

Capsuled Comments

For those of you who could not attend: We regret that you missed meeting Mrs. Mackersie and Mrs. John M. Chenault, President of the Woman's Auxiliary to the Southern Medical Association. Doctor Alfred T. Baker and Doctor Walter E. Brown, President and President-Elect of the Oklahoma State Medical Association, were two other speakers you unfortunately missed hearing.

Outstanding were the "Paris in the Spring" Luncheon and Style Show, with the charm and authentic atmosphere of a Parisian boulevard; "Spring Recital," theme of Post Convention School of Instruction, full of music and introductions; followed that evening by a fitting finale—a cocktail party and dinner-dance in the Persian Room. The State Auxiliary pays tribute once more to Mrs. C. F. Foster, Jr., Convention Chairman, and to her committee for a beautifully-planned and smoothly-run 1960 convention!

Annual Opportunity

State convention is a time of summary and a time of evaluation, and both "times" are extremely important. Progress of committee work and officers' reports can be followed at executive board meetings. But State convention is climactic in finality. Every member present feels innate pride in the work of her own county auxiliary; but the deep-seated pride and interest shared with other counties in their accomplishments is the lasting satisfaction that remains long after our meetings convene.

Organization and Membership

Since the work of every group can obviously not be covered, we feel that our upward surge in organization and membership should be presented again, particularly for those who did not hear the report. Mrs. T. A. Ragan and Mrs. Milton L. Berg, our 1st and 2nd Vice-Presidents, with Mrs. Clifford M. Bassett, President, and many members involved, are responsible for this fine record:

State Medical Association Dues-Paying Membership:	1568
State Auxiliary Membership:	1195
Differential:	373
Organized Auxiliaries in Oklahoma:	40
Unorganized Counties in Oklahoma:	5
Counties with 100% Membership:	27
Members-at-large:	26
Counties with less than 5 Doctors:	6
Auxiliaries organized this year: (Cookson Hills and Custer Counties)	2

New Officers

We are sorry if you were not present to tell Mrs. Bassett and all of our 1959-60 officers how much we appreciate their great service to us this year. If you missed meeting our new officers for 1960-61, we can all tell them now how very proud we are of them—and extend both our congratulations and our cooperation for this year:

President—Mrs. Virgil Ray Forester
President-Elect—Mrs. Pat Fite, Sr.
1st Vice-President—Mrs. Milton L. Berg
2nd Vice-President—Mrs. T. A. Ragan
Secretary—Mrs. Charles W. Freeman
Treasurer—Mrs. John W. Records
Treasurer-Elect—Mrs. Louis S. Frank
Corresponding Secretary—Mrs. Harrell Dodson, Jr.
Parliamentarian—Mrs. Clifford M. Bassett
Historian—Mrs. Charles A. Smith
Editor—Mrs. Samuel T. Moore
Publicity—Mrs. John Powers Wolff

Deaths

WILSON H. LANE, M.D.
1879-1960

Wilson H. Lane, M.D., a practicing Oklahoma physician since early statehood, died in Oklahoma City on May 15, 1960.

A native of Double Springs, Alabama, Doctor Lane graduated from Loyola University in Chicago in 1913. After practicing in Bonham, Texas for a short time, he came to Ada, Oklahoma where he practiced for 25 years before moving to Okahoma City where he and his wife, Marie Thaxton Lane, M.D., established their clinic in Britton.

Doctor Lane had served as President of the Pontotoc County Medical Society and was a member of the American Academy of General Practice and the Oklahoma Chapter of the American Academy of General Practice.

In 1956, Doctor Lane was honored for his years of service to the profession when the Oklahoma State Medical Association presented him with a Life Membership.

J. L. WHARTON, M.D.
1873-1960

J. L. Wharton, M.D., retired Depew physician, died in Bristow, May 4, 1960.

Born in Russelville, Arkansas, July, 1873, Doctor Wharton graduated from the Memphis Hospital Medical College in 1904. He established his practice in Duncan, later moving to Ketchum, Oklahoma. In 1930, he moved to Depew where he practiced until 1954. He was then associated with the Oklahoma State Veterans' Hospital in Sulphur until his retirement in 1957.

In recognition of his years of service to the medical profession, the Oklahoma State Medical Association presented Doctor Wharton with an Honorary-Life Membership in 1951.

Book Reviews

SURGERY OF THE FOOT, by Henri L. DuVries. Published by the C. V. Mosby Co., St. Louis Mo., 1959, pp. 494, price $12.50.

This is an interesting volume that touches on many of the medical and surgical problems of the foot. Despite our increasingly sedentary society, many people still use their feet. In contradistinction to the hand, few physicians have devoted most of their professional life to the foot. DuVries has done this and has contributed a volume comparable to Kanavel's early volume on the hand. The bibliographies are excellent. The written content reflects almost completely one man's experience. The Chapter on Amputations, though rightfully last, is incomplete. Illustrations are profuse.—*John A. Schilling, M.D.*

BREAST CANCER: The Second Biennal Louisiana Cancer Conference. Editor: Albert Segaloff. Published by the C. V. Mosby Co., St. Louis, Mo., 1958, pp. 257, price $5.00.

This volume includes the following sections: Basic Biology, Definitive Treatment, and Hormonal Therapy. It contains discussions and papers by widely known contributors on the various divisions of the above sections. This volume should be of value to the family physician in his decisions concerning the best form of therapy for his individual patient with breast cancer. It is a fine volume for the student of this disease as it contains the current opinions on the multiple facets of breast cancer with concluding concise bibliographies.—*John A. Schilling, M.D.*

25 YEARS AGO



Articles published in *The Journal* of the Oklahoma State Medical Association, June, 1935.

YOUR BROTHER AND YOU

Louis H. Ritzhaupt, B.A., M.D., Guthrie
President's Address
Oklahoma State Medical Association

You who are lukewarm to the affairs of government should awaken at least to the realization that the first interest of our American government is not to compel people to do certain things and restrain them from doing other things, but it is to make the right life and the useful life the natural and easy one to live.

In this country of ours the average citizen must devote a good deal of intelligent, constructive thought and undivided time to the affairs of the state as a whole, or those affairs will go backward. We cannot decline to go through all ordinary duties of citizenship for a long space of time and then suddenly get up and feel very angry about something or somebody, not clearly defined, and demand reform, as if it were a concrete substance to be handed out forthwith.

We must keep clearly before our minds the fact that brilliant ability or unusual genius play only a small part in government. It is important that the average man shall have in him the root of righteous living, a desire to help a weaker brother, and while not in any way losing his power of individual initiative, to cultivate without ceasing to further power of acting in combination with his fellows for a common end of social uplifting and of good government.

For weal or woe we are knit together and we shall go up or down together; we are all given the same qualities of heart and brain and hand which have made this republic what it is today; the same spirit will make it infinitely greater tomorrow.

The present condition of the universe was brought about by the inability of man to show restraint and subordinate individual passions and desires for the general good of the state or nation. Those who mean well, but feebly rise to action, will be of no help in the unending strife for civic betterment. Pardon can be granted to the man who has had no chance in life if he does but little for the state, but upon you who are so well qualified and who have had so many advantages rests a heavy burden to show that you are worthy of what you have received and the distinction that you cherish. A double responsibility is upon you to use aright the talents you have, not merely along professional lines, but along lines of philanthropy and public policies. Quoting the words of President Roosevelt in his last nation-wide address, "More and more of the burden of government must be placed on those who are able to pay, both in finances and in intelligence."

'60 N R A Conference Set for Oklahoma City

Outstanding national authorities in the field of rehabilitation will address the 1960 Conference of the National Rehabilitation Association, scheduled for October 10, 11, 12, at Oklahoma City's Biltmore Hotel.

As reported by Conference Chairman Don Davis, Oklahoma City, the conference theme will be "Realistic Rehabilitation Dollars and Sense." One of the highlights of the program will be the Employers' Luncheon, where some 700 employers will hear national speakers discuss employment of the handicapped.

Noted speakers appearing on the program are: Mary Switzer, Director, Office of Rehabilitation, Washington, D. C.; Sumner G. Whittier, Administrator of Veterans Affairs, Washington, D. C.; Elizabeth E. May, Dean, School of Home Economics, University of Connecticut; A. Ray Dawson, M.D., Director of Physical Medicine and Rehabilitation, V. A. Hospital, Richmond, Virginia; Doctor Kenneth Hamilton, Ohio State University; Willis Gorthy, President, Conference of Rehabilitation Centers, and Director, Institute for the Crippled and Disabled of New York; W. B. Gaines, President, Council of State Agencies for the Blind, Atlanta, Georgia; Doctor Nellie Z. Thompson, Director of Work Shop Standards; Doctor Frederick A. Whitehouse, American Heart Association, New York; Gerald Bradley, Garrett Corporation, Los Angeles, California; Justin Johnson, Hughes Aircraft, Culver City, California; and Kieffer Davis, M.D., Medical Director, Phillips 66, Bartlesville.

PHYSICIAN PLACEMENT

Dermatology

Lealand L. Clark, M.D., 200 1st St., S.W., Rochester, Minnesota; age 28; married; Columbia University College Physicians and Surgeons 1956; Board eligible July 1960; Fellows' Assoc., Mayo Foundation; Military service will be completed Sept. 1962; desires group or associate practice; available July 1960.

Joseph William Pidgeon, M.D., 5333 Littlebow Road, Rolling Hills, California; age 41; married; George Washington University 1951; Board eligible June 1960: Active Air Force Reserves; available July 1960.

General Practice

Johnny Bill Delashaw, U.S.P.H.S. Hospital, Wyman Park Drive and 31st Street, Baltimore 11, Maryland, age 27, married, University of Texas Medical Branch, 1959, available July 1960.

Robert G. Bissell, M.D., 1143 S. Wheeling, Tulsa, Oklahoma, age 28, married; University of Oklahoma School of Medicine, 1959; no military obligations; available July 1, 1960.

Robert Goodman, M.D., 63 Old Gate Lane, Powers Lake, North Dakota; age 56; married; University of Manitoba, 1930; no military obligations; available anytime 1960.

Sherman Allen Hope, M.D., 1105 Yale Ave., Panama City, Florida, PO 3-6656, age 27, married, University of Oklahoma School of Medicine, 1957, available July 1960.

Carle H. Schroff, M.D., 3811 State Line, Kansas City 11, Missouri: Age 31; married; University of Kansas, 1959; veteran; available August, 1960.

Internal Medicine

Richard I. Hochman, M.D., 37, Badger Road, Annapolis, Maryland, 31, married, New York University, 1952, available October 1, 1960.

Edward John Hertko, M.D., 2117 S. 3rd Ave., Maywood, Illinois; age 30; married; University of Illinois, 1954; no military obligations; available October 1960.

Grant Gwinup, M.D., 1121 Maiden Lane, Ann Arbor, Michigan: Age 30; married; University of Colorado, 1956; Board eligible; specialty, endocrinology; veteran; desires group practice or academic position; available July 1, 1960.

Joseph Chester Maternowski, M.D., 319 Highlake, Ann Arbor, Michigan; age 35; married; St. Louis University, 1953; eligible board of Internal Medicine & Allergy; veteran; specialty, Internal Medicine & Allergy; available July 1, 1960.

Thomas Turner Walton, Jr., M.D., 7375 Churchill, Detroit, Michigan: Age 29; married; Baylor College of Medicine, 1955; Board eligible; veteran; specialty, internal medicine; desires group or associate practice; eligible, July 1961.

Locum Tenens

Harold W. Calhoon, M.D., Wesley Hospital, Oklahoma City. One year residency in Internal Medicine. Available July 1 to September 15th, 1960.

Arnold Giesbrecht, M.D., Hallock, Minnesota, age 34, married, graduate Winnipeg, Manitoba, 1957, prefers suburban or locality close to cities.

James D. Green, M.D., second year resident in Internal Medicine at St. John's Hospital, Tulsa, desires two weeks' tenure between now and July 1. Write to St. John's or call RIverside 3-3059.

(Name on Request), O.U. graduate 1956; M.D. from Washington University School of Medicine 1959; desires locum tenen in the Tulsa area from July 1, 1960 until middle of September, 1960. Address inquiries to Key H, The Journal, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

Charles L. Maimbourg, M.D., Campus Apartments No. 5, Elf Street, Durham, North Carolina; Oklahoma University School of Medicine 1958; desires General Practice and/or Anesthesia during first two or three weeks of July 1960.

Russell M. Preston, M.D., 476 San Juan, Oak Harbor, Washington, desires six month's work in General Practice between time of separation from military service to beginning of residency training, January-July, 1960.

Obstetrics and Gynecology

Edmond Michael Brophy, M.D., 1823 Portsmouth, Westchester Illinois; married; University of Illinois, 1946; Board eligible upon completion of practice requirements; no military obligations; available July, 1960.

William Edgar Carlisle, M.D., 634 E. Patton Ave., Montgomery, Alabama; age 29; married; Tulane University School of Medicine 1953; Jr. Fellow American College of Ob-Gyn; interested in associate or group practice; available July 1960.

Joseph William Herbert, M.D., 2701 N. Azelea St., Victoria, Texas; age 40; married; Southwestern Medical School, 1945; Fellow American College of Ob-Gyn; desires associate or solo practice; no military obligations; available March 1960.

David H. Holmes, M.D., 1017 Grovena Drive, St. Louis, Missouri, 37, married, Washington University, St. Louis, Veteran, available July 1, 1960.

Gerald R. Keilson, M.D., Medical Arts Building, Dallas, Texas, age 31, married, University of Texas, 1953, board qualified, veteran, available in July 1960.

Nejdat Mulla, M.D., 1110 Belmont Avenue, Youngstown, Ohio, age 36, married, University of Geneva, Switzerland, 1952, American College of OB-GYN, not eligible military service, available July 1, 1960.

Oklahoma physician; age 35; married; Medical College of Georgia, 1954; 3 years residency training Ob-Gyn; veteran; available in 30 days. Write Key "I", Oklahoma State Medical Association, P.O. Box 9696, Shartel Station, Oklahoma City, Oklahoma.

Harold G. Ray, M.D., 212 Darien Place, Box 503, Balboa Heights, Canal Zone, age 28, married, University Arkansas School of Medicine, 1955, now in active reserve, available July 1, 1960.

Juan C. Ruiz, M.D., 350 Park Ave., Lexington, Kentucky; age 36; married; University of San Marcos Medical School 1952; no military obligations; available July 1, 1960.

Pediatrics

H. M. McClintock, M.D., 5712 St. John St., Kansas City, Missouri, 29, married, Baylor University, 1955, veteran, available December 1960.

Ophthalmology

Howard A. Dinsdale, M.D., 4624 Briarfield Road, Columbia, South Carolina; age 29; married; University of Nebraska 1954; Board Certified; available October 11, 1960.

Ira H. Kaufman, 75 S. Middle Neck Road, Great Neck, New York; age 31; married; Cornell University 1953; Board certified; desires group or associate practice; available summer 1960.

Orthopedics

Newsom Stool, M.D., c/o Mayo Clinic, Rochester, Minnesota; age 30; married; Baylor University 1953; Board eligible; available July 1960.

Radiology

Wendell M. Burns, M.D., 1158 Belvoir Avenue, Dayton, Ohio: Age 34; married; University of Louisville, 1954; Board Certified; veteran; available September 1, 1960.

Surgery

(Name on Request) 32 years old, married, Tulane, 1952, veteran, board eligible.

Clarence I. Britt, M.D., 1650 Neil Avenue, Apt 15, Columbus, Ohio; age 32; married; Ohio University College of Medicine, 1951; American Board of Surgery 1959; eligible American Board of Thoracic Surgery, 1960; inactive reserve; available July 1960.

Delbert H. McGinnis, M.D., Box 75, Clinton-Sherman AFB, Oklahoma; age 28; married; end military obligation July 3, 1960; interested in general practice; available July 4, 1960.

Duane M. Clement, M.D., 310 Bates, Boulder, Colorado; age 39; married; University of Michigan 1944; Board certified; member F. A. Coller Surgical Society; desires group or associate practice; available April 1960.

Alice F. Gambill, M.D., 1601 Clover Lane, Fort Worth, Texas; age 36; married; Oklahoma University School of Medicine 1949; member AAGP; desires general or group practice; available May 1960.

Jack T. Rush, M.D., Colorado State Hospital, Pueblo, Colorado: Age 49; married; University of Colorado, 1939; Board certified; veteran; available immediately.

Donal Wray Steph, M.D., 8519 Craighill, Dallas, Texas; age 30; married; Southwestern Medical School 1953; board eligible July 1960; available July 1960.

Clinton K. Higgins, M.D., 215 Bacon Ave., Naval Base, Norfolk, Virginia; age 62; married; Washington University 1927; Board Certified; desires administration, college health physician, group association; available May 1960.

Urology

Alex Grossman, M.D., V.A. Center, Temple, Texas, 42, married, University of Texas, 1951, board certified, veteran, available now.

John A. Malley, M.D., 3404 Sheffield Ave., Los Angeles, California; age 32; married; Marquette University 1955; Board certified; no military obligations; available July 1, 1960.

MISCELLANEOUS ADVERTISEMENTS

FOR LEASE OR RENT: Completely equipped for general practice, 3-room clinic, 26 S.W. 25th, Oklahoma City. Seven years old. Air conditioned, central heat. Will rent with or without furniture and equipment. Contact Mrs. Zelma King, 222½ S.W. 25th, Oklahoma City. CE 2-2575 or ME 2-6811.

WANTED: Young General Practitioner, without military obligations, to become partner in established clinic and hospital practice. $1000.00 per month to start. Contact Neumann-Ottis Clinic, Okarche, Oklahoma.

BOARD CERTIFIED or board eligible internist and pediatrician are needed for the Broadway Clinic, Shawnee, Oklahoma; $12,000 per year to start, with a future partnership available. Shawnee has a population of approximately 30,000, and is located within 40 miles of Oklahoma City, and the University of Oklahoma Medical Center. The Clinic owns a 40-bed, modern, air-conditioned hospital which is located adjacent to the clinic building.

WANTED: Internist to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

WANTED: Surgeon to join old established group in town of 10,000 population in West Texas. Need not be Board certified but with adequate residency training. Write Key A, THE JOURNAL, Oklahoma State Medical Association, P.O. Box 9696, Oklahoma City, Oklahoma.

FOR SALE: Office with six rooms, small laboratory, two rest rooms, storage room, air conditioned. Equipped for E.E.N.T. Near hospital. Nicely finished, with cement, cork covered floors. Good location. Equipment includes specialist's chair with stand. Phoropter, projector and space eikonometer. Special trial case, giant scopes. Ritter unit, Diatherma, Two Violet Ray units, instruments, tables, chairs, etc. Contact Edward M. Loyd, M.D., P.O. Box 2061, Harlingen, Texas.

FOR SALE: Examining room furniture and equipment. Call WI 6-1883, after 5:00 p.m.

Proceedings . . .

(Continued from Page 454)

who would serve for a period of one year. Doctor Frances B. Newlin of Shawnee nominated Clinton Gallaher, M.D., of Shawnee. There were no further nominations.

Nominations were declared in order for the office of Vice-President. Doctor C. C. Young of Shawnee nominated J. Hoyle Carlock, M.D., of Ardmore. There were no further nominations.

Doctor Gallaher called for nominations for the office of Delegate to the American Medical Association (two year term of office). The Speaker advised the House that the term of office for the Delegate and Alternate to be elected at this meeting would not commence until January 1, 1961, and that the present incumbents would serve until that time. The incumbents are Wilkie D. Hoover, M.D., Tulsa (Delegate) and Joe L. Duer, M.D., Woodward (Alternate). Following this announcement the Speaker called for nominations.

Doctor Wendell L. Smith of Tulsa nominated Wilkie D. Hoover, M.D., Tulsa, for the office of Delegate to the AMA. There were no further nominations.

The Speaker called for nominations for the office of Alternate Delegate to the AMA.

Doctor Wayne A. Starkey of Altus nominated Joe L. Duer, M.D., of Woodward, for the office of Alternate Delegate to the AMA. There were no further nominations.

The Speaker called for nominations for the office of Secretary-Treasurer. Doctor Louis H. Ritzhaupt of Guthrie nominated Johnny A. Blue, M.D., of Oklahoma City. There were no further nominations.

The Speaker called for nominations for the office of Speaker of the House.

Doctor Robert T. Sturm of Oklahoma City nominated Marshall O. Hart, M.D., of Tulsa. There were no further nominations.

The Speaker called for nominations for the office of Vice-Speaker of the House.

Doctor William T. Gill of Ada nominated C. M. Hodgson, M.D., of Kingfisher. There were no further nominations.

Next on the agenda was the nomination of Councilors for Districts Numbers 2, 5, 8, 11, 14 and 12.

DISTRICT NUMBER 2. A. M. Evans, M.D., Perry and G. B. Gathers, M.D., of Stillwater, as Councilors.

DISTRICT NUMBER 5. Ross Deputy, M.D., Clinton and C. Riley Strong, M.D., El Reno, as Councilors.

DISTRICT NUMBER 8. Wendell Smith, M.D., Tulsa and Earl M. Lusk, M.D., Tulsa, as Councilors.

DISTRICT NUMBER 11. W. A. Hyde, M.D., Durant and Thomas E. Rhea, M.D., Idabel as Councilors.

DISTRICT NUMBER 14. J. B. Hollis, M.D., Mangum and R. R. Hannas, M.D., Sentinel, as Councilors.

DISTRICT NUMBER 12. Ennis M. Gullatt, M.D., Ada (to fill the unexpired term of William T. Gill. M.D. of Ada, until 1961).

Doctor Gallaher reminded the House of Delegates members that a Councilor would also have to be nominated from Council District Number 3 to fill the vacancy created if C. M. Hodgson, M.D. of Kingfisher were elected to the office of Vice-Speaker of the House, since he could not hold two offices. He advised the Delegates from Councilor District Number 3 to hold a caucus before election of officers began.

Doctor Gallaher took this opportunity to introduce Mr. Dick Nelson, Field Representative of the American Medical Association, and Mr. Nelson gave a brief report on the status of federal legislation effecting the medical profession and some suggestions for combatting Forand-type legislation.

Doctor Gallaher announced the next Annual Meeting would be in Tulsa on May 7th through 10th, 1961, at the Mayo Hotel.

As the next order of business, Doctor Gallaher asked for a report from Wilkie D. Hoover, M.D., Delegate to the American Medical Association. Doctor Hoover gave a report on the AMA meeting held in Dallas in December, 1959. He told the House of Delegates how successful the Hospitality Room was and he also told them the American Medical Association reaffirmed their position that doctors should not be employed by hospitals because either doctor or patient is usually exploited. The AMA still stands for complete freedom of choice for the doctor, he reported.

The Speaker next called on Malcom Phelps, M.D., Delegate to the American Medical Association. Doctor Phelps gave a report on the American Medical Association House of Delegates meeting held in Atlantic City in June, 1959. Doctor Phelps said the highlight of the meeting was when the President of the United States appeared at the meeting.

Doctor Gallaher then called on Joe L. Duer, M.D., Alternate Delegate to the American Medical Association. Doctor Duer reported on the American Medical Association House of Delegates meetings in Dallas and Atlantic City. Immediately following his trip to Atlantic City he attended a breakfast in Washington, D.C., where old-age problems were discussed. Doctor Duer told the House he wanted to quote Toby Morris on what was happening in Washington today, "somebody says we have to take care of the old people, they do not ask if they need it."

Doctor R. Q. Goodwin, Alternate Delegate to the American Medical Association gave the final report on the AMA meeting in Dallas and the final action taken by the House of Delegates. There were three points stressed in the report:

1. Free Choice of Physicians—the AMA believes it is the right of each individual to choose the school he attends as well as where he starts to practice. (Sub-amendment: the AMA fully subscribes to the freedom of the physician to provide optional medical care.)

2. Physician-Hospital Relationship — A physician should not dispose of his professional services to a hospital for a fee. The practice of pathology, radiology, etc. is an integral part of the practice of medicine.

3. Veterans Administration and Socialized Medicine —Resolved: That a strong protest be made to the head of the Veterans' Administration urging more strict requirements to govern those admitted to Veterans' Administration Hospitals.

Doctor Gallaher called for any reports from the Councilors. There were no reports.

As the next order of business, Doctor Gallaher announced that the COUNCIL REPORT would be presented by Doctor Alfred T. Baker, President of the Oklahoma State Medical Association. Doctor Baker presented the following report:

(See COUNCIL REPORT on page 474.)

Doctor John E. McDonald of Tulsa moved that the Council Report be accepted. Doctor Marshall O. Hart of Tulsa seconded the motion and it passed.

Doctor Gallaher advised that the next order of business would be reports from the association's councils.

Report of the Council on Public Health

By: Bert T. Brundage, M.D., Thomas, Chairman

(See Council on Public Health report on page 475.)

Doctor Charles E. Green of Lawton moved that the report be accepted.

Doctor Louis H. Ritzhaupt of Guthrie offered a substitute motion that the part be accepted that pertains to the proper activities of the Oklahoma State Medical Association.

Doctor E. K. Norfleet of Bristow moved that pages 3 and 4 of the report not be accepted.

At this point Doctor John W. DeVore of Oklahoma City, who is the Chairman of the fourteen fact-finding committees of the Oklahoma White House Conference on Aging, spoke at length in regard to the portion of the report which pertained to the White House Conference.

Doctor Joe L. Duer of Woodward moved that an amendment be made that the report be accepted and each item be considered individually. This motion was seconded by Doctor E. K. Norfleet of Bristow. The motion was defeated.

Doctor Louis H. Ritzhaupt of Guthrie made a motion that the report be accepted and that the part which pertains to the Oklahoma State Medical Association be approved. The motion was duly seconded and carried.

Report of the Council on Professional Education

By: R. R. Hannas, M.D., Sentinel, Chairman

(See Council on Professional Education report on page 480.)

Doctor E. K. Norfleet of Bristow moved that we accept and approve the report of the Council on Professional Education. The motion was duly seconded and passed.

At 12:25 P.M., the Speaker announced the meeting would be adjourned for lunch, and would reconvene in the Mirror Room at 1:30 P.M.

The House of Delegates reconvened at 1:30 P.M. in the Mirror Room of the Municipal Auditorium. Oklahoma City.

Doctor Gallaher advised that the Association's Council reports would continue.

Report of the Council on Public Policy

By: James W. Kelley, M.D., Tulsa, Chairman

(See Council on Public Policy report on page 481.)

Doctor Joe L. Duer of Woodward moved that the report of the Council on Public Policy be accepted. The motion was duly seconded and passed.

Report on the Council on Socio-Economic Activities

By: Mark R. Johnson, M.D., Oklahoma City, Chairman

(See Council on Socio-Economic Activities report on page 485.)

Doctor Louis H. Ritzhaupt of Guthrie moved that the House of Delegates accept the report of the Council on Socio-Economic Activities and refer it to the Council of the Oklahoma State Medical Association. The motion was duly seconded and passed.

Report of the Council on Insurance

By: Simon Pollack, M.D., Tulsa, member

(Doctor Pollack read the report in the absence of Doctor Bruce R. Hinson)

(See Council on Insurance report on page 487.)

The Speaker announced that since no recommendations were made, the House of Delegates did not need to take action unless it so desired. No motion was made.

Resolutions

Doctor Gallaher announced that the Introduction of Resolutions was the next order of business, and that they would be introduced by title only at the opening session, referred to the Resolutions Committee, and formally acted upon in the closing session of the House of Delegates. The following resolutions were introduced:

1. A resolution commending Grady F. Mathews, M.D., Commissioner of Health of the State of Oklahoma, submitted by Custer County Medical Society.

2. A resolution protesting the increase in first class postal rates, submitted by the Pittsburg County Medical Society.

3. A resolution opposing compulsory social security for physicians, submitted by the Oklahoma County Medical Society.

4. A resolution in opposition to Forand-type legislation, submitted by the Oklahoma County Medical Society.

5. A resolution to support an amendment to the constitution of Oklahoma to provide more money to support institutions of higher learning and mental institutions, submitted by the Tulsa County Medical Society.

6. A resolution urging more active participation in exerting influence on local, state and national political affairs, submitted by the Tulsa County Medical Society.

7. A resolution to re-negotiate a contract with the Federal Government in the operation of the Medicare Program, submitted by the Comanche-Cotton County Medical Society.

8. A resolution to provide loan funds for first and second year medical students at the University of Oklahoma, submitted by the Kay-Noble County Medical Society.

Doctor Gallaher further announced that resolutions could also originate in the Resolutions Committee.

The next order of business was the Introduction of Amendments to the Constitution and By-Laws.

Doctor William T. Gill, Ada, reported that there were no proposed amendments to the constitution; there were three proposed changes in the By-laws, pertaining to basic requirements for membership and termination of membership in regard to Narcotic Law Violation.

The Speaker announced that the Resolutions Committee would meet in Room 1120 of the Skirvin Hotel, and that the Constitution and By-Laws Committee would meet in Room 1016 of the Skirvin Hotel, at 3:00 P.M.

Doctor Joseph M. James of Muskogee asked to make a few remarks to the House of Delegates. He told them that in order to enhance our relationship with the general public, the medical profession should come out from seclusion and speak up about contributions to society—as the care of indigents, etc.

Doctor Gallaher advised that the last order of business in the opening session of the House of Delegates would be the reading of the Necrology Report. Doctor Gallaher turned the Chair to Doctor J. Hoyle Carlock the Vice-Speaker, for the presentation of the Necrology Report.

Doctor Carlock requested that the Delegates stand during the reading of the report.

Following the reading of the Necrology Report, the Speaker announced that the House of Delegates of the Oklahoma State Medical Association, Opening Session, would recess at 2:30 P.M. to reconvene at 5:00 P.M.

REPORTED BY DORIS LEE GEORGE

COUNCIL REPORT

The last council report had as its opening paragraph the following: "It seems rather ironical that each Council Report of the Oklahoma State Medical Association finds the profession still seeking to assist in solving problems, not only in the scientific field of medicine, but the problems also of the body of politics and those of economics." From the problems of the last twelve months it would seem that there has been no change in the situations which have confronted the Association.

This report (as those in the past) cannot possibly deal with all of these problems or recite the profession's accomplishments in the past year or years, or contemplated in the future.

These remarks of the Council are made only to draw to the attention of the House of Delegates that each delegate should weigh carefully his deliberations of the reports and recommendations that will be made by the several committees of the Association.

Membership

As of May 1, 1960, the membership of the Association was as follows:

Paid Membership	1,568
Life and Honorary Membership	125
For a total membership of	1,693
This is in comparison of a total membership at the same date in 1959, of	1,639

Finance and Budget

The Council once again reiterates that the estimating, in 1960, of the income and expenditures of the Association for the year 1961 is virtually impossible.

The House of Delegates at its last meeting approved the recommendation of the Council that the Association as rapidly as possible create a reserve fund of at least one-year's operating expense which at that time was approximately $100,000.00, but which as of this date is now approximately $120,000.00. This action of the House of Delegates is being carried out by the Council within the limits of the funds available.

On December 31, 1959, the assets of the Association were as follows:

Cash on hand	$32,243.13
Savings and Loan (Ponca City, Lawton, Bartlesville)	22,354.65
Total	$54,597.78

Since December 31, 1959, the account with the Home Savings and Loan Association of Bartlesville, has been increased to a total of $10,000.00, and an additional $10.000.00, has been placed with the Durant Building and Loan Association. Thus, the total reserve assets of the Association as of this date not counting cash in the bank is in the amount of $40,000.00, all accounts earning 4% interest.

Considering these financing factors and the present operating costs of the Association, and any capital investments that might have to be made, your Council recommends that the dues of the Association remain at $42.00, for the year 1961.

Budget

INCOME	
DUES	$69,000.00
ANNUAL MEETING	9,000.00
JOURNAL	54,000.00
MISCELLANEOUS	1,000.00
TOTAL INCOME	$133,000.00

Expenses

GENERAL ADMINISTRATIVE EXPENSE	$55,000.00
ANNUAL MEETING	16,000.00
COMMITTEES	4,000.00
TRAVEL (in and out of state)	6,000.00
JOURNAL	47,000.00
INCOME OVER EXPENDITURES	$ 5,000.00

Journal

The House of Delegates' attention is again called to the continued expansion of the Journal.

Last year the total number of pages of copy increased from 744 pages in 1958, to 840 pages in 1959. The pages of advertising in 1958, was 923 and increased to 1,035 in 1959.

The Council regrets that it must announce that Doctor Ben H. Nicholson, the Editor of the Journal, has found it necessary for personal reasons to resign. The Council in this report expresses to Doctor Nicholson its sincere appreciation for the excellency of his contributions to his profession and the Journal during the years he has served as editor.

Annual Meeting

In the 1959 Council Report which was adopted by the House of Delegates, the Annual Meeting city was secured three years in advance with the meeting to be held in 1961 in Tulsa. In cooperation with the Tulsa County Medical Society and the Tulsa Chamber of Commerce, the meeting dates have been set as May 7-10, 1961.

The Council commends Doctor Charles M. Bielstein, General Chairman, and his committees for the splendid work done in making arrangements for the Annual Meeting this year.

Councils

In last year's Council Report it was recommended that the committee structure of the Association be streamlined by creating five councils as follows:

A. Council on Public Health

B. Council on Public Policy

C. Council on Socio-Economic Activities

D. Council on Insurance

E. Council on Professional Education

The House of Delegates approved this procedure on a trial basis for a period of one year.

During the past year under the new operational procedure, much has been accomplished and yet at the same time there has not been sufficient experience for the Council procedure to be tested as to its efficiency and therefore, it is recommended that this type of organization for the Association be given one more year of experimentation before there is a definite recommendation as to necessary changes in the By-Laws to make the change permanent.

Honorary-Life Members

The Council has had the following applications for Honorary Life Memberships submitted to it and recommends their election:

Edgar A. Johnson, M.D., 103½ East Jackson, Hugo

Charles Nelson Berry, M.D., 1816 Drury Lane, Oklahoma City

Hull W. Butler, M.D., 1430 North West 27th, Oklahoma City

L. D. Hudson, M.D., 308 East 8th, Dewey

Clarence O. Epley, M.D., 1200 North Walker, Oklahoma City

Walter A. Huber, M.D., 2222 East 25th, Tulsa

Waldo B. Newell, Sr., Broadway Tower, Enid

Walter S. Larrabee, M.D., 416 Medical Arts Building, Tulsa

G. I. Walker, M.D., 402 North Price, Hominy

Legislation

While this subject will be presented to the House of Delegates by the Public Policy Committee, the Council, nevertheless, feels that this subject cannot be over-stressed with particular emphasis on the Forand Bill (H. R. 4700) now before the national Congress.

Unless each member of the medical profession pulls his share of the load, final enactment may be inevitable.

With regard to medical and hospital care for either the medically indigent or persons over 65, this House of Delegates is reminded that at a special-called session of the House of Delegates held February 21, 1960, the House of Delegates approved the plan of Blue Cross-Blue Shield "Special 60 Program" for individuals over 60 years of age. The result of the enrollment of persons over 60, under this program is not known at this date. The Council urges the profession to give careful consideration to this program of Blue Cross-Blue Shield to the end that it shall not be abused.

The House of Delegates' attention is also directed to the Smathers-Morton-Keogh-Simpson Bill (H. R. 10) which will allow self-employed persons to create their own retirement. This bill, now before the Senate Finance Committee, is in the process of being reported out of Committee, and Senator Robert S. Kerr of Oklahoma, as the second ranking member of the Senate Finance Committee, is in a strategic position for its final enactment. As has been reported previously in the "Osma-scope", the profession should let Senator Kerr know their feelings in this matter.

With regard to state legislation, this will be dealt with in the report of Public Policy Committee.

White House Conference

As reported in the last Council report, there were to be two White House Conferences, one in 1960 and one in 1961,—one on youth and the other on aging. Reports on both will subsequently be a part of the Council on Public Health.

It is obvious that the White House Conference on Aging may have far reaching import on the ultimate type, if any, of federal legislation for the medically indigent, or aged.

Industrial Commission

During the past months there has been a great deal of publicity concerning the manner of operation of the Industrial Commission and the manner in which medical testimony was given. Officers of the Association and members of the Grievance Committee have attended all legislative hearings on this matter and as of this date no member of the Industrial Commission or the Legislative Council has seen fit to make public the names of the accused physicians.

The Association and the Board of Medical Examiners appeared before the Legislative Council and pledged their cooperation not only to the Legislative Council but to the Industrial Commission in any and all respects that might in any way clear up the existing situation.

The Oklahoma Bar Association offered its services in re-writing the Industrial Commission Law and it is assumed and probable that the Association will assist the Bar Association in this endeavor.

COUNCIL ON PUBLIC HEALTH

Bert T. Brundage, M.D., Chairman

The Council on Public Health at the beginning of the year was composed of the following committees:

Aging, School Health, Rehabilitation, Perinatal and Maternal Mortality, Mental Health, Cancer, and Civil Defense.

In the latter part of this year, on request of the Chairman of the Council, a Liaison Committee to the Public Health Department was appointed, the Chairman of this Committee being Doctor Paul Erwin of Oklahoma City. This latter Committee was appointed to survey the State of Oklahoma with regard to the medical supervision of county health units as directed by the previous House of Delegates.

A questionnaire was created and sent to all county and/or district medical societies for a report on all 77 counties of the State of Oklahoma.

The survey brought reports from 43 of the 77 counties. A review of the surveys showed that only 7 county medical societies had ever met with county health officers to discuss the public health problems of their respective counties. The survey also developed these points with regard to the present operation of county health units:

1. County health units treating patients free, who are able to pay.

2. Non-cooperation from physicians in private practice.

3. The majority of the counties would like to have a fulltime public health unit. The survey further showed that there was probably insufficient medical supervision of the activities of the county health departments in the majority of counties where there was not a fulltime public health unit.

Predicated upon the results of this survey, the Committee addressed a letter to the Commissioner of Health, Grady F. Mathews, M.D., asking as to whether or not the Oklahoma State Medical Association could assist the Department in securing better medical supervision of county health departments where indicated. Doctor Mathews immediately replied to the effect that the Department would welcome such cooperation and it is the hope of the Liaison Committee that there will be such cooperation between the State Association and the Department of Public Health.

Committee on School Health

Robert D. Shuttee, M.D., Chairman

The activity of the Committee on School Health was limited to participation in the White House Conference on Children and Youth.

The White House Conference on Children and Youth began in 1909, when the first meeting was called by President Theodore Roosevelt. The conference has been held every 10 years since and concerns itself with the past, present, and future needs of the children and youth of our Nation.

The theme of the 1960 Conference was: "To promote opportunities for children and youth to live a full and creative life in freedom and dignity." Over 7,000 attended the meeting, 14% of whom were represented by the Youth. All organizations and individuals throughout the Nation, interested in the health and welfare of children, were represented. There were five general theme assemblies, eighteen forums, and two hundred ten work groups. Each individual was assigned to a specific work group, so that each topic would be represented by various professions and organizations.

In view of the fact that there were a limited number of representatives from Oklahoma to attend these forums. it was necessary that the Oklahoma representatives determine the most important workshop they would attend. On the basis of this determination, your representative elected to attend the forum on "The Young with the Social Handicap;" and the work group topic was, "Neglected Children." This proved to be a very interesting work group and seemed to offer several positive steps toward aiding our children and youth to lead a more useful life. Though the need for the physically handicapped was recognized, our discussion was directed to the fact that more attention is needed for the early recognition and therapy for the emotionally handicapped. It was shown that the physician, caring for children, plays an important role in detecting and directing therapy for the emotionally disturbed child.

A system was proposed for state and community commissions, represented by various professions and organizations to direct attention to all phases in the care of neglected children.

All in all, your representative was personally impressed with the Conference. As one physician said, before the meeting started, that he was in a state of psychological blank; your reporter was of the same opinion until the purpose was unfolded. Obviously, with such a large number represented, there were some members present who were not satisfied with their work group topics, but, if an adequate follow-up of even a few of the suggestions will be made on a national, state, and above all, a local level, the meeting was a success.

Committee on Aging

J. Walker Morledge, M.D., Chairman

Background

The activities of the 1958-59 Committee on Aging more or less set the course for its successor's efforts during the past year, when it initiated state action in regard to the White House Conference on Aging. Because of this special carry-over project, all but one of the Committee's regular activities were suspended in favor of working on the Oklahoma White House Conference on Aging. The Sub-Committee on Rehabilitation and Restoration of the Aged continued its activities, and a summary of its accomplishments is included in this report, along with a review of White House Conference Activities.

Oklahoma White House Conference on Aging

The previous committee had just begun its work of determining the health needs and status of Oklahoma's

aging and aged populations, when it was learned that Congress had enacted legislation, P. L. 85-908 calling for a national White House Conference on Aging, scheduled to be held in Washington, D. C., in 1961. A further provision of the law authorized federal grants of $5,000-$15,000 to states, the purpose of which was to help the states to finance local conferences during 1960.

On the assumption that the available Federal grant would be sought after by various state agencies engaged in the provision of health care, the committee became interested in seeing to it that the funds and the Oklahoma conference were managed by an unbiased agency of state government. For this reason, the previous committee recommended that the Governor apply for the funds on behalf of the Extension Division at the University of Oklahoma. The Governor granted this request.

The manner in which the statewide study has been handled to date, indicates that the previous committee was wise in its recommendation. All but one state has applied for the Federal money, but few of the state activities are being operated with the degree of impartiality that is apparently being displayed in Oklahoma.

Activities of the Present Committee

Due to the tremendous scope of the statewide study for the Oklahoma White House Conference on Aging and the fortunate involvement of committee members and other OSMA members in this study, the Committee on Aging membership has been absorbed into the committee structure of the White House Conference undertaking rather than continue all of its previous activities. More than sixty Oklahoma physicians are participating in key positions: OSMA President Doctor Alfred T. Baker, is a member of the Governor's Committee; Doctor John DeVore is chairman of all the fourteen fact-finding committees which are being employed in in the study; the Chairman of your Committee on Aging is serving as chairman of the Fact Finding Committee on Health of the Aged; and Doctor Lee Honska is Chairman of the Committee on Research, Training and Demonstration. Mr. Blair is on the committee which will make recommendations as to the selection of delegates to the national conference.

Other physicians who have participated have done so in various positions throughout thirteen of the fourteen fact-finding committees; through service on and/or chairmanship of mayor's committees in Oklahoma cities of over 2500 population; and, through appearances on the programs of regional conferences held in key areas of the state. While physicians involved in conference activities have of necessity been operating as individuals, they have tried to reflect the philosophy of organized medicine in every possible way.

The results of this gathering of information and exchange of ideas will be reported to the Oklahoma White House Conference which is scheduled to be held in Norman on June 7, 8 and 9, 1960. From the conference certain recommendations will be made concerning the health status of Oklahoma's senior citizens, and these recommendations will be transmitted to the national White House Conference on aging which will be held in Washington, D. C., in January, 1961.

Health Fact Finding Committee

Since physicians have been primarily responsible for the preparation of the report of the Health Fact Finding Committee, a brief summary of the organization of this committee and its recommendations follow:

The major committee was sub-divided into three areas of investigations: Health Care Resources, Health Care Needs and Health Care Financing. After a careful review of the above subjects and their application to the increasingly large aging and aged segments of the Oklahoma population, the following recommendations were made:

Public Health Resources

1. Joint development of a continuing intraprofessional (medical) campaign focused on developing and maintaining joint and united action for improvement of state and local health department's staff and program.

2. A joint and continuing epidemiologic type of survey of the public's use and demand for services, as a measure of effective need.

3. It is recommended that the medical profession join efforts with the county officials to obtain state legislative appropriations of monies, for allocation to the counties, in an amount of 50¢/capita. This would provide 1.1 million dollars compared to $385,358 appropriated for 1960.

4. In order that the Oklahoma State Department of Health be able to meet its statutory and other public health obligations toward all age groups at a minimal acceptable level, it is recommended that similar efforts be made to obtain legislative appropriation of a similar amount ($1.1 million) for the operation of the State Department of Health. (Costs have increased approximately 43% in 10 years while state appropriations have increased by about only 15%.)

Dietetic and Nutrition Resources

1. Increase full time dietitians services by 39 dietitians.

2. Provide 24 dietitians to share their services among geographically convenient hospitals.

3. Provide six nutrition consultants to be strategically located in the various areas, plus one chief nutritionist; all under direction and supervision from the State Department of Health, thus enlarging its nutrition unit.

4. Also, place a senior dietary consultant under the supervision of the Chief Nutritionist at the State De-

partment of Health and one consultant each to be assigned to areas A and B.

The objectives are to provide dietetic services to all hospitals and nursing homes, and for the other public health aspects of nutrition through education, economic surveys of food and nutrition costs, etc.

5. In order to achieve the above recommendations, the necessity of more qualified nutrition educators and expanded educational programs must be recognized. Therefore we recommend that funds and opportunity be made available to provide advanced training to develop nutrition educators.

6. We recommend that a continuing program be developed and directed by qualified instructors for the training of food service personnel in all institutional types of medical and domiciliary care facilities.

7. Studies need to be made to determine the nutritional status and dietary practices of the aged and other age groups in Oklahoma; both in their homes and in all institutional types of medical and domiciliary care facilities.

Nursing Resources

1. Encourage better utilization of graduate nurses by developing flexible work schedules patterned to the needs of married nurses and recent unmarried graduates.

2. A continuing program for the redevelopment of nursing skills in the temporarily retired graduate nurse whose diminishing family responsibilities will allow her to return to active nursing.

3. The development and organization of a continuing nurse aide training program under the sponsorship of an educational agency, with cooperation from other health agencies, in order to take advantage of the findings and materials developed through the pilot project of the State Department of Health.

4. Inform physicians, communities, and their leaders regarding the visiting nursing services that can be developed and provided through their county health units, to fit the needs of the particular physician's patient.

Health Care Financing

1. No major legislative action should be taken until the completion of the National White House Conference on Aging, January, 1961.

2. Oklahoma must uphold voluntary enterprise as the system of choice and oppose additional federal spending.

3. Oklahoma must conduct a comprehensive survey of the status of its senior citizens and develop corrective health programs at the family, city, county and state levels—in that order.

4. Voluntary programs should be emphasized, with local tax-supported activities limited to the truly indigent, and/or disabled who cannot be helped through voluntary means.

5. The medical care funds of the Department of Public Welfare should be re-directed to the extent that better and more nursing and domiciliary home care may be provided to the indigent.

6. Ways and means for easing the financial burden of catastrophic illness of the aged must be studied and solved at the state level. The term "catastrophic" must be clearly defined.

7. Rehabilitation of the aged should be included as a part of the creation of a central diagnostic rehabilitation facility at the state level.

8. The financial plight of the aged must be relieved through greater employment opportunities and a relaxation of income restrictions imposed upon welfare and social security recipients. Income restrictions should be flexible, to the extent that they are consistent with the economy.

9. Voluntary prepaid health plans and commercial health insurance companies must be inspired and encouraged to develop more and better programs for the aging and aged.

10. County medical societies, public health units and community leaders must establish a better liaison and spirit of cooperativeness designed to accomplish more and better voluntary programs to assist the needy elderly in the prevention of illness and the correction of health problems.

11. To coordinate the accomplishment of the above, it is recommended that the Governor establish, under his direction, a "dollar-a-year" type Committee on Aging, with a full-time Executive Director. The committee should be comprised of professional, educational, religious and business leaders of the state, with sufficient staggered tenure to permit continuity of planning. The committee must be free from politics and from the control of any agency or department of government. Similarly constituted committees should be established at the county level.

General

1. A measure of quantitative adequacy of physician-time should be developed in relation to the work to be done; along the lines of the work of Dr. Roger I. Lee, done for a federally sponsored health study in 1933. It is even more necessary now, with changes in medical and surgical skills, population shifts, costs, travel time, and the progressive shifting of the quantitative relationships between family-physician-time and specialist-time, the definition of their joint relationships to modern high level comprehensive medical care; and, most important, as a guide to judgment in these matters.

2. A local (state) health information gathering operation should be instituted on a continuing basis, and for all related health fields—physicians, dentists, hospitals, nursing homes, nurses and other para-medical personnel; also for population illness surveys, utilization of service and facilities. This data would be available to all groups contributing to it. The use of data other than that contributed from a given group would be available for use and/or publication only on a joint or a mutually agreeable basis.

The management of any segment of the health field by the members of that segment in relation to the other segments is just as handicapped by lack of information on trends and current status and follow-up as would be the management of any business-like operation.

It is suggested that such a health information center be located and developed in the University of Oklahoma Medical Center, under the supervision and operating responsibilities of the Biostatistical Section of the Department of Preventive Medicine and Public Health.

Since many of the above recommendations will undoubtedly be argued at the state conference and since the final outcome of the state conference will have a direct bearing upon the Oklahoma recommendations to be transmitted to Washington, the Committee on Aging has planned to meet with representatives of the Oklahoma Hospital Association, the Oklahoma Nursing Home Association and the Oklahoma State Dental Association, prior to the conference, in order that the interested health professions will be adequately represented during conference activities.

It is our understanding that the conference will receive wide advance publicity and that individual invitations will be mailed to members of the medical profession. Your committee therefore *recommends* that all of you make plans to attend the conference in the interest of adequate professional representation.

Committee on Rehabilitation

Earl D. McBride, M.D., Chairman

The first meeting of the Committee on Rehabilitation of the Oklahoma State Medical Association was held on November 15, 1959 at 2:00 p.m.

Resolutions were adopted by the Committee as follows:

1. "Definition of rehabilitation is the restoration of the individual to a functional capacity; mentally, physically, and psychologically; to establish as nearly as possible an independent status in life."

2. "A medical diagnostic rehabilitation facility be established at the University Medical Center to which disabled individuals could, by proper referral, be evaluated en toto through a team approach of all medical specialists and co-medical personnel to determine and coordinate the individual's rehabilitation objective and treatment with the necessary cooperation of all private and public groups and agencies that might be so concerned."

The second meeting of the Committee was held on January 10, 1960 at 2:00 p.m. Further resolutions were adopted as follows:

"In an effort to enhance the training of physicians of the State of Oklahoma, it is recommended that a Diagnostic Rehabilitation Center be organized and coordinated for research and teaching at the University of Oklahoma School of Medicine. At this center, disabled individuals could, by proper referral, be evaluated en toto through a team approach of all medical specialists and co-medical personnel to determine and coordinate the individual's rehabilitation objective and treatment with the necessary cooperation of all private and public groups and agencies that might be so concerned."

The following resolution was also approved by the Committee:

"We, the Committee on Rehabilitation of the Oklahoma State Medical Association, do resolve that: It is a worthy and profitable endeavor for us, as citizens of the United States, to endorse, promote, and assist in the rehabilitation of physically and emotionally handicapped persons so that they may become occupationally and vocationally restored and self-supporting.

"And we find that we can endorse the current philosophy of rehabilitation as practiced by the Oklahoma State Department of Vocational Rehabilitation in:

"1. Using practicing physicians in the local communities to evaluate its clients (on a fee for service basis), and;

"2. Relying upon practical medical judgment to select and treat those cases (on a fee for service basis) which, in the light of present medical and educational knowledge and abilities, are actually feasible for vocational rehabilitation, and;

"3. Employing available persons in private, charitable, and public institutions, and placement services to disabled persons, to accomplish their vocational rehabilitation in a practical economical manner, and;

"4. Performing these acts in a manner which retains and enhances the personal sense of dignity and independence of the individual who is undergoing rehabilitation.

"And, we therefore resolve to commend the present administration of the Oklahoma Department of Vocational Rehabilitation for its efficient performance in accordance with the American principles of achievement that strengthen the individual; and we urge that this work continue and be expanded in accordance with these principles and objectives."

Another resolution was adopted in which it was noted that the National Rehabilitation Association would hold its meeting in Oklahoma City in October, 1960. A motion was made and passed that the Rehabilitation Committee promote and present a Scientific Exhibit at this meeting entitled "The Role That Physicians Play in Rehabilitation of the Handicapped." The object of the exhibit would be to have a number of physicians and specialists present who would answer questions of the social workers and other members attending the convention, also some statistics showing the number of examinations and surgical operations and medical treatments, and other statistics showing the services rendered by physicians in the field of rehabilitation in Oklahoma.

Three exhibit spaces for the NRA meeting are being made available on a complimentary basis. The planning of the exhibit has been delegated to a sub-committee which is chaired by William K. Ishmael, M.D.

This Committee for 1960 recommends that at least some continuity be used in appointments for 1961.

Sub-Committee on Rehabilitation and Restoration of the Aged

Herbert Kent, M.D., Chairman

As previously stated, this sub-committee continued to function despite the conflicting activities of the White House Conference.

The group submitted a preliminary report of its activities in April of 1959, but has now completed its studies related to the problem of rehabilitation and restoration of the aging and aged.

Because of the inherent complex nature of the aging problem and the multi-faceted solutions which are possible for rehabilitating an aging individual, the report was subdivided into five specific areas: The Problem, Prevention, Diagnosis, Treatment and Facilities.

The current status of rehabilitation is comprehensively reviewed in the report and recommendations are made for increased interest and activity in this vital feature of the health care of the aged.

The committee observes that there is a great need to restore the physical, mental, social, vocational and economic life of the senior citizen. It is further stated that the magnitude of the task and the necessity for an enlightened attitude toward the needs of the aging ill can be successfully met by recognizing the importance of the individual rather than of his disability.

Other Committees

Other committees of the Council have not filed a report.

COUNCIL ON PROFESSIONAL EDUCATION

R. R. Hannas, M.D., Chairman

The Council on Professional Education has consisted of a chairman and three committees:

Post Graduate Education
Medical School Liaison
American Medical Education Fund

The report of each of these committees follows:

Post Graduate Education Committee

(1) This committee has been engaged in organizing a series of post-graduate medical education courses to be presented throughout the State at locations most easily accessible to the physicians of all areas in an attempt to bring continuing medical education as close to home as possible for our members. It is believed that such a program will (1) provide a more enlightened physician for the people of the State of Oklahoma, (2) strengthen the bonds between the practicing physician and the State Association, and (3) perhaps decrease malpractice claims through the educational benefits provided. Accordingly, the following recommendation is made:

Recommendation

That a Decentralized Post Graduate Medical Education Program shall be sponsored by the Oklahoma State Medical Association in conjunction with the University of Oklahoma Medical School, to begin as soon as practicable, and have the following provisions:

(a) Faculty for the courses to be furnished by the Medical School, and to be paid a suitable fee plus travel expenses.

(b) Dates and sites for the courses to be determined by representatives of this committee working in conjunction with the Executive Office of the Oklahoma State Medical Association, the Medical School, and physicians in the areas concerned.

(c) The number of courses presented to be determined by local physicians' reception and support.

(d) Each course generally to consist of three hours of lecture and one hour of discussion, the lectures providing basic science, research, and clinical coverage of a particular organ-system, to be presented when practicable at an evening dinner meeting.

(e) Courses to be financed by tuition fees charged attending physicians, with the Oklahoma State Medical Association underwriting any financial losses incurred not to exceed $2,500.

(2) In an attempt to foster progressive postgraduate medical education, a second project has been under consideration by this committee. This concerns a postgraduate seminar to be presented at the University of Oklahoma Medical Center and would involve an entirely new method of conducting a postgraduate course. It is believed that in sponsoring such a course the Oklahoma State Medical Association may make an outstanding contribution to the field of postgraduate medical education, which may serve as a model for other states or schools to follow. Details of the plan are as follows:

(a) Enrollment would be limited to 30 physicians.

(b) The sessions would last for 2½ days, tentatively beginning on Thursday morning and finishing Saturday at noon.

(c) The group of 30 would be divided into five groups of six each.

(d) Each half day session would last from 2½ to 3 hours.

(e) Each group of six would meet with three instructors, a local staff man serving as moderator, a local faculty member particularly skilled in the topic to be discussed, and a guest discussant from one of the nearby medical schools.

(f) The participants would be required to supply questions on each topic in advance, these would then be assembled, mimeographed, and distributed to all discussants and participants in advance.

(g) The seminars would be truly round table, informal, and no lectures as such would be given.

(h) Each participant would spend half a day with each group of discussants and thereby cover five topics during the 2½ day period.

(i) Outline for scheduling would be as follows, with the five groups of physicians indicated as A, B, C, D, E, and the five groups of topics by numbers:

Topic	Thur AM	Thur PM	Fri AM	Fri PM	Sat AM
1	A	B	C	D	E
2	B	C	D	E	A
3	C	D	E	A	B
4	D	E	A	B	C
5	E	A	B	C	D

(j) If more than 30 applicants apply, those not selected for the first course would receive preference for a repeat course, either the same year or the following year, the number of courses to be determined by the number of applicants.

(k) If more than 30 applicants apply, selection will be made geographically so that no one area, such as Oklahoma City, would receive preference.

Recommendation

That the Oklahoma State Medical Association in conjunction with the University of Oklahoma Medical School, sponsor the above seminar, sponsorship, in this instance, meaning to underwrite this course for an amount not to exceed $500.

Medical School Liaison Committee

The Medical School Liaison Committee assisted the Postgraduate Education Committee in working out the details of the above mentioned postgraduate courses. Cooperation by the Medical School representatives was excellent and it is believed that relations between the School and the Association have been strengthened in this regard.

American Medical Education Fund

Though no formal committee has been appointed, the activities of this Association in support of the AMEF have been bequeathed to this Council. Suffice it to say that although no specific campaign was held this year on a state-wide basis, through the efforts of individual physicians and the Women's Auxiliary, the total contributions from this Association reached a new high, with $2,478.06 contributed from 95 donors as contrasted to $1,602.50 from 66 donors the year before. In the correspondence files of this Council are plans which may be utilized to provide more active support to the AMEF in the coming year.

Recommendation

That a formal committee be appointed for the purpose of continuing the support of this worthy fund.

COUNCIL ON PUBLIC POLICY

James W. Kelley, M.D., Chairman

State Legislative Committee

During the past year, the activities of the Council have been primarily devoted to the field of federal legislation, since there has been no occasion for meetings of the State Legislative Committee, inasmuch as the Oklahoma State Legislature has not been in session. However, it is contemplated at the moment that the State Legislative Committee will recommend two measures to the next Legislature: (1) A Medical Examiner System to replace the County Coroner System in unexplained deaths. (2) A bill that would relieve the physician of public liability when he serves in an emergency manner on persons other than his own patients.

Grievance Committee

The Grievance Committee has continued its work, but during the past year there have been only five cases for its consideration and, with the possible exception of one case, the committee believes that its services were satisfactory.

Federal Legislative Committee

Obviously the past year has been devoted principally to Forand-type legislation. The Forand Bill (H.R. 4700) was refused favorable consideration by the House Ways and Means Committee by a vote of 17-8. Following this action, information was received that Senator Smathers of Florida was considering introducing compromise legislation whereby the Federal Government would also participate in medical and hospital care for the needy. According to the best information available, the plan would ask that three changes be made in regulations concerning the use of federal matching funds in state medical care programs.

The Changes Are As Follows:

1. Federal matching funds for state medical care programs (such as the one currently operated in Okla-

homa by the Department of Public Welfare) should not be limited to categories of the needy sick, such as the blind or aged, but should be available to be used in providing medical, hospital and nursing home care, and drugs for all those in need of the care, as locally determined, without the means of providing it for themselves.

2. Federal matching funds for state medical care programs for the needy sick should be appropriated and administered at the federal and state levels separate from funds used for subsistence programs. The practicing medical profession should have a responsible position in the planning and management of these medical programs—which can best be done through state agencies that are under the direction of medical doctors.

3. The local, or county, governments should be permitted to voluntarily participate in the matching fund program to provide medical care for the needy sick, just as states may voluntarily participate in the federal matching program. Such a system would encourage community interest and responsibility in providing this care. Thus, states will be encouraged to develop their individual programs for hospital and medical care of the needy sick, rather than creating new, national legislation which would not be tailored to local needs.

It is reported that the philosophy behind such a proposal was that Washington observers felt that some sort of national legislation is inevitable, and that the Smathers plan would be more palatable than a medical care program for the aged which would be affixed to the Social Security System.

After much discussion the Federal Legislative Committee, at a recent meeting, approved *the following recommendation:* "If an established need for additional federal health care for persons over 65 is determined, then the following principles are recommended: That the qualified aged of Oklahoma, as determined by an effective, enforceable means test, receive health care, on a voluntary basis by counties, through a program to be administered by the Oklahoma State Health Department and by the medical profession."

Of additional interest to the profession is the Smathers-Morton-Keogh-Simpson Bill (H.R. 10). This bill has passed the House of Representatives and is now in the Senate Finance Committee, where Senator Robert S. Kerr is the second ranking member. Senator Kerr has recently surveyed several professional groups in Oklahoma as to their attitude to this proposal and it is believed that Senator Kerr will be helpful in attempting to bring the bill before the Senate for a vote.

A new development has also presented itself with regard to compulsory social security for physicians. The House Ways and Means Committee has recently proposed that physicians be included under the Social Security Act.

The American Medical Association House of Delegates and the vast majority of state medical societies have historically opposed participation in social insurance plans and your Council believes that physicians of Oklahoma are still opposed to being brought into the program on a compulsory basis.

Some general observations concerning trends in government show that there appears to be grave danger of the United States bankrupting itself within the next decade, particularly if the present trend in spending continues.

Much of the criticism leveled at the medical profession is a result of the effects of inflation, and we are but an incidental target of an irate public that is getting squeezed in the inflationary whirl.

As an example, the real villain of the elderly is inflation; inflation brought about by the United States Congress and its fiscal irresponsibility. Now, Congress is looking for a scapegoat and we have been elected.

The purchase of medical care through additional taxation will only temporarily relieve the symptoms of the impoverished aged; it will not only fail to correct the cause, but will provide momentum to deficit spending and its concomitant, worthless money.

In attempting to solve all of the "social ills" of the country through additional legislation, Washington continues to make matters worse. It must be recognized, before it is too late, that the frantic pursuit of brush fires, as evidenced by Forand-type legislation, will never reverse basic trends. And the inflationary trend today is one of dire forebodings for Americans of all ages, and, indeed, for the future of our nation.

Physicians have a moral obligation to keep themselves well-informed in governmental affairs, and a responsibility to assume roles of leadership in returning government to the people.

Your Council hopes that the coming year will show evidence of even greater effort on the part of the medical profession to discharge these responsibilities and obligations.

Osteopathic Relations

Your Council also had one other matter to come before it and this problem concerned a request from the Oklahoma State Osteopathic Association for a meeting to review inter-professional relations. The Council agreed that it would honor this request only if the Osteopathic Association would furnish an agenda well in advance of the meeting in order that the problems to be studied could be known. This action of the Council has been communicated to the Osteopathic Association but no definite date has as yet been selected for the meeting nor has the Council received an agenda. It should be quite obvious to all that the Council would take no action without referring its ultimate recommendations to the House of Delegates.

Federal Legislative Committee

J. R. Stacy, M.D., Chairman

During the past year, the Federal Legislative Committee, consisting of Doctors J. Hoyle Carlock, Tom

Gafford, Clinton Gallaher, Worth Gross, Bill Harsha and Paul Lingenfelter, has been quite active in its efforts to inform Oklahoma physicians and others regarding key legislation of particular significance to the medical profession.

It was first necessary to achieve effective organization, so that the individuals, the cities and the counties would be well-informed and their opinions well-represented in the nationwide network of coordinated activity which was being sought by the Field Service Division of the American Medical Association.

The organization was basically developed by the geographic distribution of committee members, to the extent that each physician on the state committee represented one of Oklahoma's Congressional Districts. Each physician on the committee was armed with a Legislative Notebook and placed on the American Medical Association's mailing list for follow-up information.

The individual members of the committee, in turn, established their own network of communication throughout the counties of their respective Congressional Districts. Not only were their activities to be directed toward members of the profession, but they were also encouraged to solicit the interest and support of other business and professional groups, as well as from individuals of all walks of life. Ideally, the broad objective was to use medical leadership to arouse the public as a whole in the affairs of American Government, to the end that some degree of representative government might be reclaimed from planners at the federal level.

The committee accomplished moderate success in its ambitious undertaking.

Among the federal bills receiving special attention during the past year were the Forand Bill (H.R. 4700), and the Smathers-Mroton-Keogh-Simpson Bill (H.R. 10).

Forand Bill

The appeal for tax-paid medical care for the aged, as represented by the Forand Bill, has fulminated during this session of Congress, even more so than during the closing days of the preceding Congress last summer.

On both occasions of crisis when the House Ways and Means Committee held public hearings on H.R. 4700 (July, 1959 and February, 1960), the committee was extremely successful in marshalling hundreds of letters to Washington. The results of such typical reaction on a national scale brought about the demise of the Forand Bill both times; the last time by a 17-8 vote of the House Ways and Means Committee.

In addition to disseminating information and soliciting support through personal correspondence, the OSMA-Scope and the Journal, numerous meetings have been coordinated by members of the Federal Legislative Committee; Oklahoma's Congressional Delegation was hosted at breakfast in Washington, D. C. last spring; physicians of the First Congressional District have had encouraging meetings with Representative Page Belcher; Representative Toby Morris of the Sixth Congressional District has met with physicians and others in Lawton; and, informal and formal meetings have been held with other Representatives and Senators throughout the year.

Members of the committee have made scores of speeches before a great variety of groups over the State. Much support for our position against the Forand Bill has been achieved in this laborious manner.

The Federal Legislative Committee also held an important meeting with the leaders of other state organizations such as the Farm Bureau, insurance associations, related health organizations and other business and professional groups. Many of the organizations present responded by initiating activities of their own in combatting Forand-type legislation.

Committee members have been unusually successful in receiving editorial support from several key newspapers over the state, including the metropolitan press of Tulsa and Oklahoma City.

In summary, Oklahomans have played an important role in defeating the Forand Bill twice during the last year.

Despite moderate effectiveness, the threat of Forand-type legislation is still imminent. Most presidential candidates are on record as favoring some sort of bill providing health care for the aged, and it appears that all candidates will develop such proposals in the near future. The Democratic Party has endorsed the principles of providing a legislative solution for furnishing health care to the aged, and the Republicans are wavering in their support of voluntary enterprise as the system of choice.

The pressure of the A.F. of L.-C.I.O. and the political expedience of courting the old folks during an election year portend a continuing fight ahead. It is virtually assured that strong efforts will be made by both parties to at least achieve compromise legislation during this session of Congress.

At an Easter Sunday meeting of the Federal Legislative Committee, an informal proposal by Senator Smathers of Florida was read and discussed. It was reported that the Florida State Medical Association had tentatively approved the Smathers plan.

According to the best information available, the plan would ask that three changes be made in regulations concerning the use of federal matching funds in state medical care programs.

1. Federal matching funds for state medical care programs (such as the one currently operated in Oklahoma by the Department of Public Welfare) should not be limited to categories of the needy sick, such as the blind or aged, but should be available to be used in providing medical, hospital and nursing home care, and drugs for all those in need of the care, as locally determined, without the means of providing it for themselves.

2. Federal matching funds for state medical care programs for the needy sick should be appropriated and administered at the federal and state levels separate from funds used for subsistence programs. The practicing medical profession should have a responsible position in the planning and management of these medical programs—which can best be done through state agencies that are under the direction of medical doctors.

3. The local, or county governments should be permitted to voluntarily participate in the matching fund program to provide medical care for the needy sick, just as states may voluntarily participate in the federal matching program. Such a system would encourage community interest and responsibility in providing this care. Thus, states will be encouraged to develop their individual programs for hospital and medical care of the needy sick, rather than creating new, national legislation which would not be tailored to local needs.

It was reported that the philosophy behind such a proposal was that Washington observers felt that some sort of national legislation is inevitable, and that the Smathers plan would be more palatable than a medical care program for the aged which would be affixed to the Social Security System.

After much discussion the Federal Legislative Committee approved the following recommendation: "If an established need for additional federal health care for persons over 65 is determined, then the following principles are recommended:

"That the qualified aged of Oklahoma, as determined by an effective, enforceable means test, receive health care, on a voluntary basis by counties, through a program to be administered by the Oklahoma State Health Department and by the medical profession."

Smathers-Morton-Keogh-Simpson Bill

Commonly referred to as the Keogh Bill, this nine-year-old effort to achieve tax equity for the self-employed is enjoying a good chance for passage, although it is quite likely that it will be modified to some extent.

As originally proposed, the Keogh Bill (H.R. 10) would allow for the self-employed to defer income tax on an annual set-aside of $2,500, or 10% of income, whichever is less, up to a lifetime maximum of $50,000.

The Treasury Department has reportedly objected on the basis that such liberal treatment for the self-employed is in itself discriminatory. Efforts are being made at the national level now to effect a modification that will satisfy the Treasury Department without neutralizing the intent of the bill.

Your committee has repeatedly appealed to the membership for letters to their Representatives and Senators in support of this bill. Senator Kerr, the second-ranking member of the Senate Finance Committee, wherein the bill now rests, has recently surveyed several professional groups in the state to test their attitudes toward the original legislation, as well as toward the compromise.

A continuing effort must be made to get this bill out of committee and to the floor of the Senate.

Compulsory Social Security

A recent action by the House Ways and Means Committee purposes compulsory inclusion of physicians under the Social Security Act. The Administration had previously recommended that physicians be covered by the program.

The American Medical Association House of Delegates and the vast majority of state medical societies have historically opposed participation in social insurance. In addition, there is a growing awareness outside the profession, among businessmen and other conservative elements of our nation, of the socialistic nature and inequities of the Social Security program.

Although some older physicians would personally profit by participting in this scheme, it would only occur at the expense of coming generations. Younger physicians could obtain two to four times the security provided by Social Security through voluntary, private investments of many sorts.

Actually, the older age group of physicians would benefit very little when it is considered that over 50% of physicians who retire do so after the age of 74. A survey shows that more than 85% of the doctors between the ages of 65 and 72 are in active practice and could not draw benefits because their annual incomes would exceed the maximum allowable.

Your committee believes that physicians are sufficiently intelligent to more adequately provide for their own security.

General Observations Concerning Trends in Government

There appears to be grave danger of the United States bankrupting itself within the next decade, particularly if the present trend in spending continues at the present rate.

Much of the criticism leveled at the medical profes sion is a result of the effects of inflation, and we are but an incidental target of an irate public that is getting squeezed in the inflationary whirl.

As an example, the real villain of the elderly is inflation; inflation brought about by the United States Congress and its fiscal irresponsibility. Now Congress is looking for a scapegoat, and we have been elected.

The purchase of medical care through additional taxation will only temporarily relieve the symptoms of the impoverished aged; it will not only fail to correct the cause, but will provide momentum to deficit spending and its concomitant, worthless money.

In attempting to solve all of the "social ills" of the country through additional legislation, Washington continues to make matters worse. It must be recognized, before it is too late, that the frantic pursuit of brush fires, as evidenced by Forand-type legislation, will never reverse basic trends. And the inflationary trend today is one of dire forebodings for Americans of all ages, and, indeed, for the future of our nation.

Physicians have a moral obligation to keep themselves well-informed in governmental affairs, and a responsibility to assume roles of leadership in returning government to the people.

Your committee hopes that the coming year will show evidence of even greater effort on the part of the medical profession to discharge these responsibilities and obligations.

REPORT OF THE COUNCIL ON SOCIO-ECONOMIC ACTIVITIES

Mark R. Johnson, M.D., Chairman

This Council, established by the House of Delegates at its annual convention, April 1959, consists of the following committees:

Committee on Prepaid Medical Care, Henry T. Russell, M.D., Chairman

Committee on Labor and Miscellaneous Third Party Programs, Joe L. Duer, M.D., Chairman

Committee on Occupational Health, Charles M. O'Leary, M.D., Chairman

Committee on Medical Care Program for Public Welfare Recipients, Mark R. Johnson, M.D., Chairman

A single, short, sad and tired word forms the basis for this report: Apathy.

As constituted, the Council held no meeting of the various committee chairmen. The Council chairman did not call a meeting, and had he done so perhaps the attendance would have been 100 percent—perhaps. The chairman failed to call a meeting because he was—well, busy; busy practicing medicine; busy attending meetings; busy raising a family; busy paying taxes; busy writing a thousand reports and more. He didn't call one because he was tired. Tired in many ways, but also tired of trying. Tired of trying to impose another meeting, and another trip on his tired and busy colleagues. Undoubtedly the most important reason that no meeting was called is simply no meeting was required. It was something that could be put off until tomorrow and all the other hundred things had to be done today. Pressure produces apathy, too. It becomes a sort of defense mechanism, resistance against further pressure. Passive resistance true, but resistance.

The chairman of the Committee on Prepaid Medical Care cites a rather alarming example of apathy in our association. Utilization rates are spiraling along with unit costs in the "Blue Plans." In an attempt to get some ideas concerning the cause of the over-utilization, the Committee wrote letters to the presidents of all county societies where an increased utilization had appeared in the past year. They were requested to comment and give their thoughts as to why utilization was increasing in their areas. Result? Not a single reply. Not even an acknowledgment of the letter. Thus it is recommended that a special committee be formed to study the causes of over-utilization of pre-paid medical care plans. It is recommended that such committee should be authorized to study the Blue Cross and Blue Shield records if such is agreeable by that organization.

Dr. Duer reports for the Committee on Labor and Miscellaneous Programs and observes that "organized" medicine seems to be making no headway whatsoever against organized labor. Who has even seen an apathetic union organizer? Dr. Duer states that his committee has observed a need for complete re-evaluation of the individual and collective policies of the medical profession. Specifically this should be directed toward achieving uniformity in our relationships with "third party" health programs in general. Here then, a consolidation is recommended in contrast to the separation cited above.

More prime examples of apathy come from the Welfare Medical Care Committee. Last year each county society was asked to poll its membership and express its favor or disfavor of the establishment of a "Central Trust" which could receive contributions from physicians who did not want payment for services rendered welfare patients. Two replies were received. Since the institution of the Medical Care Program, physicians have been urged to enter their "usual fees" in the appropriate spaces on welfare claims. This information is of vital importance both to us and our profession. Knowledge of such sums would make it possible now to say how much free medical care was rendered in this program. Result? So few entries that they are statistically without merit and thus we have no valid reply to the ingrate who asks, "well you got paid fer it didn't you?"

Perhaps it is time someone comes up with a suggestion to strengthen and preserve our own Association. As a committee can serve no purpose without meeting, an association cannot survive without attendance. Isn't it somewhat appalling to realize that it would be possible to elect a State President who has never attended a single county or state meeting? Perhaps there should be attendance requirements as a prerequisite for nomination. Perhaps there should be a nominating committee which would examine qualifications and add some order to the selection and announcement of candidates. Perhaps, too, there should be a requirement that each component society send minutes of its meeting together with signed attendance rosters to the state association. Isn't it somewhat frightening to realize that we ask our president, our spokesman, our public representative to forsake his practice without remuneration, to abandon his family without comfort and to espouse our interests without support? Contrast this picture with the leaders, our adversaries: the labor unions, the bureaucrats, the welfare trust. Perhaps we should see to it that an honorarium of real substance be awarded to our elected president in order that he may fight our battles and strengthen our organization in a much more direct and dignified way. And perhaps we should hire some help not quite so tired, not quite so busy, not quite so apathetic. People can write letters, make speeches, consolidate committees, prepare reports, evaluate statistics; it is quite apparent that practicing physicians cannot. Do we need to do something? Will we do anything?

Report of Committee on Medical Care For Recipients of Public Welfare Assistance

April 15, 1959 through April 15, 1960

Committee Organization

Mark R. Johnson, M.D., Chairman, Oklahoma City
George H. Garrison, M.D., Oklahoma City
E. M. Gullatt, M.D., Ada
Thomas E. Rhea, M.D., Idabel
Richard Burgtorf, M.D., Shattuck
E. H. Shuller, M.D., McAlester

Formal committee meetings were held on two occasions, June 17, 1959, and November 12, 1959. The committee was duly constituted, and a quorum was present at each meeting. The elected chairman presided.

At the end of its second full year of operation, the Welfare Medical Care Program faces looming financial difficulties. From July 1, 1959 through February 29, 1960, slightly more than $4,100,000 was paid on hospital and physicians claims. Additional claims for nursing care, rendered both in nursing homes and in recipients' own homes, for ambulance services and blood bank payments, have contributed to the total cost of $9,862,000 during the eight month period. Approximately 120,000 claims were paid with these funds. During a similar period of time in 1958-1959, the program had cost $9,700,000 in payments on 140,000 claims. Payments to physicians have averaged $50.23 per claim compared to $50.80 per claim last year. During the current report period, the average hospital stay was 8.45 days compared with 9.4 days last year. All of this seems to speak well for the group and individual efforts made to direct and regulate the program. Nursing care costs have continued to rise, however, as the population of this recipient category increases. The statistical clue to the mystery of the financial difficulties is found in the number of claims honored and the number of recipients carried on the rolls, eligible for the benefits of the medical care program. This is shrinking and will continue to do so, gradually decreasing the total income to the program. Simply stated, when the number of paying policy holders decreases and the underwriting cost continues unchanged, the day of bankruptcy is forthcoming.

Commonly, financial difficulties can be resolved in two ways; by increasing the income or decreasing the expenditure. Since the medical care program operates on a fairly fixed income, formula (fluctuating with the energy or lethargy of business and sales tax revenue) virtually nothing can or should be done in an effort to pirate a greater income. Consequently, attention is now being directed to some curtailment of expenditures, specifically in the physician-hospital category. (All of us have been and will continue to be urged to promote the soundness and economy of the program by cooperating with the spirit of the regulations governing it. It is apparent beyond the shadow of any reasonable doubt that some physicians misunderstand, misapply, or completely disregard the limitations of a just and appropriate claim. When our Association agreed to endorse this program, it did so with a clear understanding that we would when and if necessary monitor our own membership.

Last May, two hospitals in the state were visited by teams representing the Welfare Medical Care Program, in an effort to study the causes of excessive utilization rates. Last week, in accordance with established and approved policies adopted by this Association, the first case involving physician misapplication of the program was referred from the Professional Advisory Committee to the Grievance Committee of the State Association. Thus, two slightly cumbersome and distinctly unpleasant regulatory procedures have been put into effect. Others can follow. If our own internal efforts fail, outside efforts are bound to follow, accompanied by acrimonious publicity, protests of persecution and charges of fraud. If we do not meet with success in our efforts to stay within the spirit of program, we will in shortsighted resentment revoke our endorsement, admit our failure and turn a tribute into condemnation.

During the report period certain changes have occurred in the program:

On July 1, 1959, Senate Bill 20 became effective and in substance transferred the Crippled Children's Program to the Department of Public Welfare. This transfer did not effect the fundamentals of the Medical Care Program, however, as the State Medical Association has not endorsed payment of physicians claims for services rendered children. In September certain procedures involving oral surgery were approved and added to the medical care program. On October 15, 1959, the professional advisory committee to the Department of Public Welfare meeting in regular session approved the establishment of a claim category for nursing care in the home to be rendered the adult recipients of ADC funds. In substance this provided for the payment of nurses and/or attendants who are necessary to care for either of the parents of dependent children in the event of home confining illnesses. This extension gives the parents of dependent children a program which is more nearly identical to the one provided old age recipients. The medical representatives of the Professional Advisory Committee spoke and voted against the motion, but it carried with the support of the majority. It should be pointed out that this extension will not increase the current financial problems of the program since the funds for payment of claims in this category will come from ADC premiums which are at present in surplus. Premiums received from ADC payments can by legal interpretation be expended only in the purchase of services and goods for eligible persons in that category. Thus, they cannot be transferred to the old age, blind or disabled group funds for distribution or medical care payment. In November a new form regarding need for nursing care in the recipients own home was prepared and distributed by the Department of Public Welfare. It is hoped that the proper execution of this form by recipients and attending physicians will expedite appropriate care and relieve the physician of the burden of decision. Also in

November the scope of the medical care program for adult recipients in the ADC category was broadened to permit treatment which might reasonably be expected to restore such persons to a state of health, which would lead toward self care, self support, and the maintenance and strengthening of the family life; ultimately designed to shorten the ADC rolls. It may be of interest to note that a pilot study has been carried out in 1958 and 1959, resulting directly in the removal of 244 cases in slightly less than 12 months. The closure of these cases earlier than the average two and one-half years dependency saved the Department approximately $800,000. Consequently, it is believed that this extension of the medical care program is, to paraphrase, pound wise and penny foolish. When the Oklahoma Public Welfare Commission initiated the pilot program for Aid to Dependent Children's parents in November 1958, it established the regulation which states: "Eligibility will not be considered established unless the incapacitated parent accepts responsibility for carrying through any treatment prescribed by the examining physician, at whatever point such treatment becomes available to him, if the treatment is such that a reasonably prudent man would accept, and may be expected to effect the clients capacity for self-support." This refusal to accept treatment may automatically start procedures to remove that person from the rolls.

Your state committee on Medical Care for welfare recipients was advised of the action taken by the Professional Advisory Committee at its meeting on November 12, 1959. It unanimously supported a motion that we record disapproval of Public Welfare payments for nursing care in the homes of the ADC recipients.

Plans are being made to establish statistical analyses in the various phases of the Medical Care Program. This project, which will create only negligible financial demand, holds great promise and should obtain the support of every participant. In this connection it is pertinent to point out that most physicians fail to insert the amounts of the usual billed charges for the services rendered welfare cases. Appropriate space is provided on the standard claim and should be utilized by every physician in the state. This feature is of the utmost importance for it is in this way and only in this way that we can, as the months go by, learn the actual amount of *free* medical care which we provide in this program. Ultimately this will be our most valuable reply to critics accusing us of mercenarism,

In reflecting upon past events and anticipating future events, it is apparent that a long road has been set upon so far, successfully. The steepest hills will be financial ones and the most important bridges will be built upon our own integrity. As we stand at this milestone with the eyes of at least a part of a Nation watching we must resolve to complete the journey. Through our participation in this program, we may recapture the support of the people and perhaps eventually climb out of the lengthening shadow of Federal medicine.

COUNCIL ON INSURANCE

Bruce R. Hinson, M.D., Chairman

The Council is made up of two Committees: namely: Professional Liability, Ralph A. Smith, M.D., Oklahoma City, Chairman, and Group Insurance, with E. C. Yeary, M.D., Ponca City, as the Chairman.

It goes without saying that at the moment the interest of the profession more probably lies in the field of Professional Liability in view of recent publicity in this field.

As an introduction to the subject of Professional Liability, your Committee would like to advise the House of Delegates that of approximately 1625 members of the Association, 1388 are participating in the Association liability program.

Professional Liability Committee

The Professional Liability Committee met with representatives of the St. Paul-Mercury Insurance Company, the insurance carrier for the Association, on April 21 for a discussion of the rate structure as it pertains to the loss ratio.

Representatives of the Company gave a detailed report of this experience, since the inception of the program in 1952, which is as follows:

PREMIUM COSTS

1952	Physicians	$52.00	Surgeons	$ 52.00
1959	Physicians	54.00	Surgeons	81.00
1960	Physicians	67.50	Surgeons	141.75
Total Premiums	Physicians	$443,409	Surgeons	$832,573
Losses	Physicians	$215,847	Surgeons	$658,169
Ratio of Losses to Premium	Physicians	.487	Surgeons	.791

The combined premium of physicians and surgeons is thus $1,275,982, and the combined losses $874,016, with an average ratio of .685.

From these figures it is quite obvious that rates for doctors of medicine who are doing surgery will increase approximately 64% at the time of their premium paying date, after the increase goes into effect, while the rate for physicians doing no surgery will remain the same.

Under these circumstances it would only be natural to inquire if professional liability insurance could be purchased cheaper elsewhere. For the information of the House of Delegates, even with this increase in rates to surgeons, the St. Paul rates are 7.5% under those of the Bureau Companies.

Your Committee and Council, after considering all of the factors involved in the Public Liability field, both in Oklahoma and the nation as a whole, feel that the efforts of the St. Paul-Mercury to cooperate with the Association in holding down and defending claims is most meritorious.

Oklahoma Medical Association Professional Liability Program

1. Inception of Program—June, 1952
2. Number of Active Members—1,625
   Number of Participating Members—1,388
   Participation—85.4%

3. Oklahoma Rate History Since Inception of Program:

$10,000-$30,000 Limits Minimum

	St. Paul		Bureau	
	Phys.	Surg.	Phys.	Surg.
(A) At Inception of Program	$52.00	$ 52.00	$50.00	$75.00
(B) St. Paul Increase June 15, 1956	54.00	81.00	50.00	75.00
(C) Increase May 9, 1957	67.50	141.75	67.00	117.00
(D) Bureau Change December 2, 1959	50.00	105.00	54.00	130.00

(E) On January 5, 1960, St. Paul Decreased Partnership Charge and Lowered Rates for X-Ray and Shock Therapy.

Present St. Paul rates are 7.5% below Bureau for Physicians and 19.2% below for surgeons.

4. Experience Exhibit

	Written Physicians Premiums	Incurred Losses	Ratio	Written Surgeons Premiums	Incurred Losses	Ratio
1952	$ 42,354	$—	—	$ 4,969	$—	—
1953	53,894	32,698	.61	36,299	1,440	.004
1954	53,766	22,290	.42	41,852	37,598	.90
1955	57,302	65,174	1.14	51,033	53,506	1.04
1956	41,383	15,924	.39	79,822	72,124	.91
1957	55,744	19,579	.35	179,044	62,338	.35
1958	59,447	25,205	.42	203,008	76,278	.375
1959	64,656	28,253	.436	192,429	115,355	.60
*1960	14,863	6,724	.452	44,117	239,530	5.43
Total	$443,409	$215,847	.487	$832,573	$658,169	.791

*3 Months

Physicians and Surgeons Combined

	Written Premiums	Incurred Losses	Ratio
1952	$ 47,323	$—	—
1953	90,193	34,138	.38
1954	95,618	59,888	.63
1955	108,335	118,680	1.10
1956	121,205	88,048	.73
1957	234,788	81,917	.35
1958	262,455	101,483	.386
1959	257,085	143,608	.558
*1960	58,980	246,254	4.18
Total	$1,275,982	$874,016	.685

*3 Months

5. Outstanding Reserve Analysis (End of Year)

Year	Oklahoma
1952	$—
1953	27,700
1954	70,950
1955	140,700
1956	160,550
1957	95,900
1958	116,200
1959	178,400
April 1 1960	$398,269

6. Proposed Rates

	Present St. Paul	Proposal St. Paul	Bureau
Physician	$ 67.50	$ 67.50	$ 72.90
Surgeon	105.00	172.80	175.50
Additional Charges:			
(1) X-Ray Therapy	50.00	50.00	54.00
(2) Shock Therapy	50.00	50.00	54.00
(3) Employed Physicians	12.50	12.50	13.50
(4) Employed Surgeons	26.50	32.00	32.50
(5) Employed Laboratory Technicians	5.00	5.00	5.00
(6) Employed X-Ray Therapy Technicians	13.00	13.00	13.00
(7) Partnership Liability	20%	20%	20%
(8) X-Ray Therapy by Employed Physician and Surgeon	12.50	12.50	13.50
(9) Shock Therapy by Employed Physician and Surgeon	12.50	12.50	13.50

Group Insurance Committee

The Group Insurance Committee supervises the Health and Accident and Life Programs.

Both of these programs have operated most successfully. The Life Group, with the Massachusetts Mutual Life Insurance Company, has been in effect since 1956. During this time the Trust has collected **$254,599.95** in premiums and paid out 14 claims in the amount of **$225,000.00.** The program covers **461** members of the Association. During the initial enrollment period all members under 65 years of age were eligible without evidence of insurability. This provision is true now for all new members if application is made within 31 days after becoming a member. Any member under age 65 is eligible at any time upon evidence of insurability.

The Health and Accident Program has a truly outstanding record for its first year of operation. The report for the first year given to the Committee by the insurance carrier, The North American Company, shows that **966** members are enrolled in the program. Premiums collected amounted to **$116,684.71,** and paid out in claims, **$28,801.39.** If this excellent experience can be continued for a year or two, it is quite obvious that there can be either a reduction in premium or an extension of benefit.

There has been inquiry made by some members concerning an Association program for overhead expense coverage. This field will be explored by the Committee in the near future.

for adults and children



Prompt 4 way check of diarrhea

- Curbs excessive peristalsis
- Adsorbs toxins and gases
- Soothes inflamed mucosa
- Provides intestinal antisepsis

POMALIN

TRADEMARK

Liquid

EFFECTIVE ANTIDIARRHEAL

FORMULA: *Each 15 cc. (tablespoon) contains:*

Sulfaguanidine	2 Gm.
Pectin	225 mg.
Kaolin	3 Gm.
Opium tincture	0.08 cc.
(equivalent to 2 cc. paregoric)	

DOSAGE: Adults: Initially 1 or 2 tablespoons from four to six times daily, or 1 or 2 teaspoons after each loose bowel movement; reduce dosage as diarrhea subsides.

Children: ½ teaspoon (=2.5 cc.) per 15 lb. of body weight every four hours day and night until stools are reduced to five daily, then every eight hours for three days.

SUPPLIED: *Bottles of 16 fl. oz. (raspberry flavor, pink color)*
Exempt Narcotic. Available on Prescription Only.

Winthrop LABORATORIES
New York 18, N. Y.



The first synthetic penicillin available for general clinical use

FOR YOUR NEXT PATIENT WHERE PENICILLIN IS INDICATED

SYN

PEAK BLOOD LEVELS HIGHER THAN POTASSIUM PENICILLIN V

ORAL ROUTE PROVIDES HIGHER INITIAL PEAK BLOOD LEVELS THAN INTRAMUSCULAR PENICILLIN G

IMPROVED ANTIBIOTIC ACTION FROM ISOMERIC COMPLEMENTARITY

SUPPLY: SYNCILLIN TABLETS—250 mg. and SYNCILLIN TABLETS—125 mg.
SYNCILLIN FOR ORAL SOLUTION—60 ml. bottles—when reconstituted, 125 mg. per 5 ml.
SYNCILLIN FOR PEDIATRIC DROPS—1.5 Gm. bottles. Calibrated dropper delivers 125 mg.





CONSIDER THESE 6 IMPORTANT THERAPEUTIC ATTRIBUTES OF

SYNCILLIN™



potassium phenethicillin (POTASSIUM PENICILLIN-152)

ANTIBIOTIC ACTIVITY DIRECTLY PROPORTIONAL TO ORAL DOSE

REDUCED RATE OF INACTIVATION BY STAPH PENICILLINASE

SOME STAPH STRAINS MORE SENSITIVE TO SYNCILLIN IN VITRO

BRISTOL LABORATORIES, SYRACUSE, NEW YORK

BRISTOL







NEOCHOLAN®

Your patient will often respond promptly to Neocholan therapy. It greatly increases the flow of thin, nonviscid bile and corrects biliary stasis by flushing the biliary system. It also relaxes intestinal spasm, resulting in an unimpeded flow of bile and pancreatic juice into the small intestine. Neocholan helps to promote proper digestion and absorption of nutrients. It also encourages normal peristalsis by restoring intestinal tone.

Each tablet provides: Dehydrocholic Acid Compound, P-M Co. 265 mg. (Dehydrocholic Acid, 250 mg.); Homatropine methylbromide 1.2 mg.; Phenobarbital 8.0 mg. Supplied in bottles of 100 tablets.

PITMAN-MOORE COMPANY
DIVISION OF ALLIED LABORATORIES, INC.
INDIANAPOLIS, INDIANA

ANOTHER YEAR OF SYMPOSIA . . .

Recognizing that the exchange of ideas is fundamental to medical progress, Lederle continues its Symposium program with the 9th year of scheduled meetings. Through these Symposia, sponsored by medical organizations with our cooperation, over 50,000 physicians have had the opportunity to hear and question authorities on important advances in clinical medicine and surgery. You have a standing invitation to attend any of these Symposia with your wife, for whom a special program is planned.



ANCHORAGE, ALASKA
Saturday, June 11, 1960
The Westward Hotel

WEST POINT, NEW YORK
Thursday, Friday, Saturday,
June 16, 17, and 18, 1960
United States Thayer Hotel

*MADISON, WISCONSIN**
Thursday, June 23, 1960
The Holiday Inn

*SPRINGFIELD, MISSOURI**
Sunday, June 26, 1960
The Holiday Inn

*ROANOKE, VIRGINIA**
Saturday, July 16, 1960
The Hotel Roanoke

*SANTA ROSA, CALIFORNIA**
Friday, September 16, 1960
The Flamingo Hotel

*KANSAS CITY, KANSAS**
Friday, September 23, 1960
Battenfeld Memorial
Auditorium

HOUSTON, TEXAS
Saturday, September 24, 1960
The Shamrock Hilton Hotel

DEFIANCE, OHIO
Wed., September 28, 1960
Defiance College

PHILADELPHIA, PENN.
Sunday, October 16, 1960
The Sheraton Hotel

*HARTFORD, CONNECTICUT**
Thursday, October 20, 1960
The Statler Hotel

*GREAT FALLS, MONTANA**
Saturday, October 22, 1960
The Rainbow Hotel

ROCHESTER, NEW YORK
Wednesday, October 26, 1960
The Manger Hotel

CHARLESTON, WEST VIRGINIA
Sunday, October 30, 1960
The Daniel Boone Hotel

SIOUX FALLS, SOUTH DAKOTA
Tuesday, November 1, 1960
The Sheraton-Cataract Hotel

*CHARLOTTE, N. CAROLINA**
Thursday, November 3, 1960
The Hotel Charlotte

*CLEVELAND, OHIO**
Wednesday, November 9, 1960
Pick Carter Hotel

*SOUTH BEND, INDIANA**
Friday, November 18, 1960
The Pick-Oliver Hotel

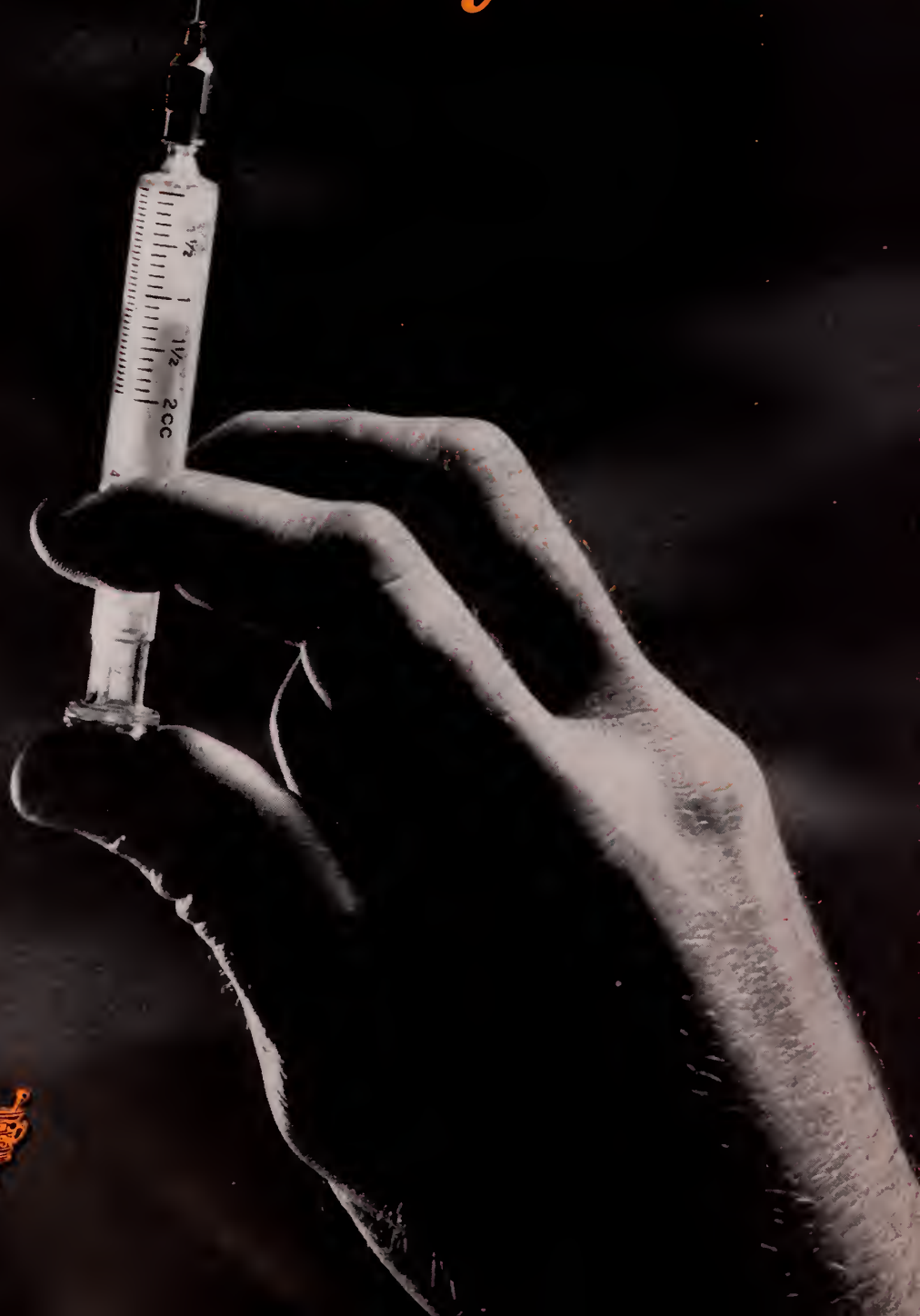
WESTCHESTER COUNTY, N. Y.
Wednesday November 30, 1960
Westchester Country Club

ST. PETERSBURG, FLORIDA
Saturday, December 3, 1960
Tides Hotel and Bath Club

*Acceptable for Category I Credit for members of American Academy of General Practice

Lederle LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, N. Y.

"life saving" in many cases...



Bristol

KANTREX®

INJECTION

Kanamycin Sulfate Injection

...a highly potent, bactericidal antibiotic for combating staph and gram negative infections

...well tolerated when used on a properly individualized dosage schedule which does not induce excessive blood levels

"In many instances its effect has been dramatic and life saving . . ."[1]

"Six of the patients who survived were considered to be terminally ill at the time kanamycin was started but showed dramatic improvement and eventual complete recovery."[2]

"... indeed, the results [with kanamycin] are the most remarkable ever achieved with otherwise fatal staphylococcal infections that we have ever seen."[3]

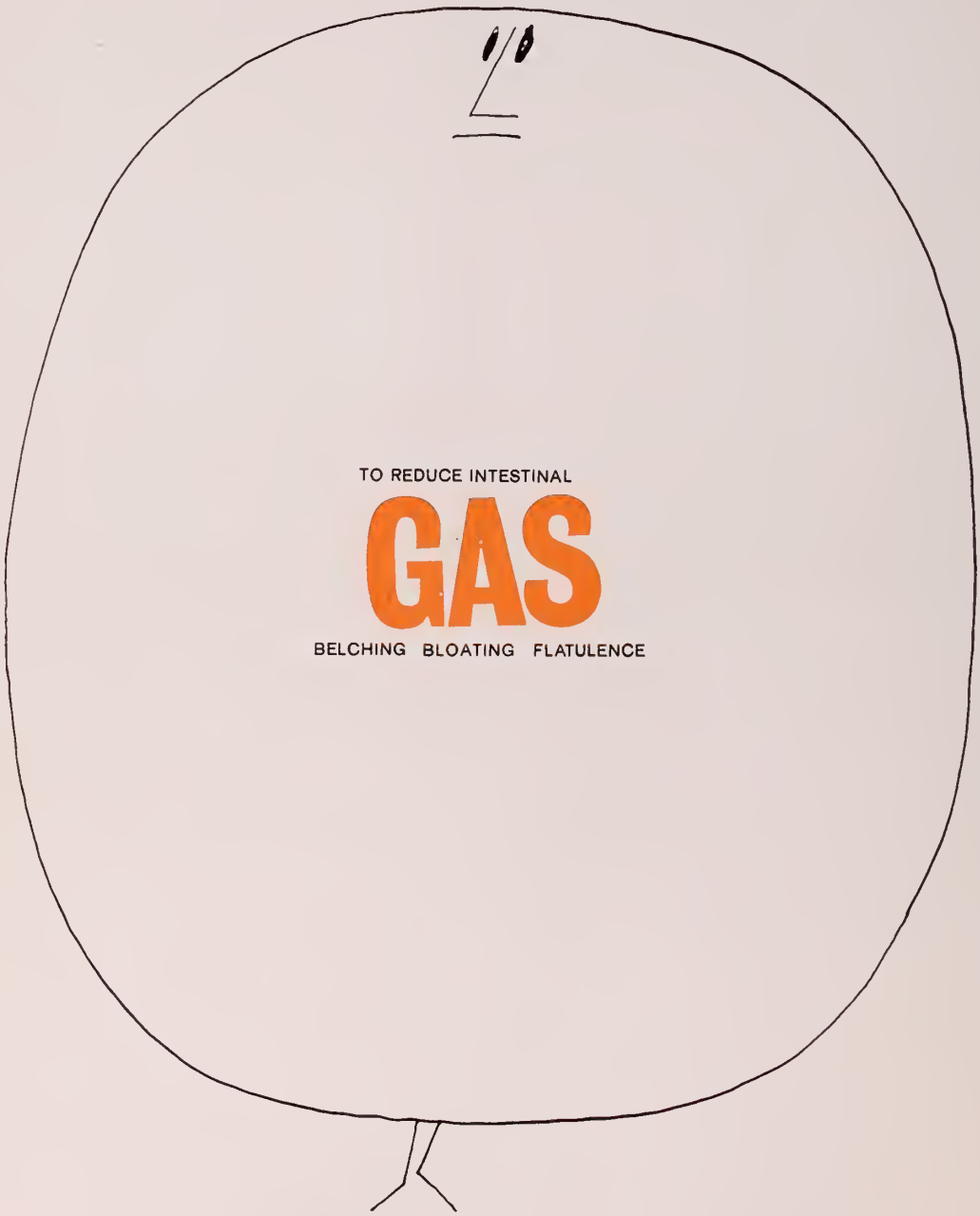
"There appears to be no doubt that kanamycin has been lifesaving in those instances in which organismal resistance precludes the use of other antimicrobials."[4]

Information on dosage, administration and precautions contained in package insert or available on request.

SUPPLY: KANTREX Injection, 0.5 Gm. kanamycin (as sulfate) in vial containing 2 ml. volume. KANTREX Injection, 1.0 Gm. kanamycin (as sulfate) in vial containing 3 ml. volume.

REFERENCES: 1. Yow, E. M.: Practitioner 182:759, 1959. 2. Yow, M. D., and Womack, G. K.: Ann. N. Y. Acad. Sci. 76:363, 1958. 3. Bunn, P. A., Baltch, A., and Krajnyak, O.: Ibid. 76:109, 1958. 4. Council on Drugs, J.A.M.A. 172:699, 1960.

BRISTOL LABORATORIES, SYRACUSE, NEW YORK



A biochemical compound used to diminish intestinal gas in healthy persons and those patients having digestive disorders ■

KANULASE

Each Kanulase tablet contains Dorase*, 320 units, combined with pepsin, N.F., 150 mg.; glutamic acid HCl, 200 mg.; pancreatin, N.F., 500 mg.; oxbile extract, 100 mg. Dosage: 1 or 2 tablets at mealtime. Supplied: Bottles of 50 tablets.

*DORSEY BRAND OF CELLULASE, EXPRESSED AS DIGESTIVE ACTIVITY UNITS

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska

For topical infections, choose a 'B. W. & Co.'® 'SPORIN'...



'CORTISPORIN'®
brand **OINTMENT**

Combines the anti-inflammatory effect of hydrocortisone with the comprehensive bactericidal action of the antibiotics.

Each gram contains:
'Acrosporin'® brand Polymyxin B Sulfate 5,000 Units
Zinc Bacitracin ..... 400 Units
Neomycin Sulfate ..... 5 mg.
Hydrocortisone ..... (1%) 10 mg.
in a special petrolatum base.



Provides comprehensive bactericidal action effective against virtually all bacteria likely to be found topically.

'NEOSPORIN'®
brand **ANTIBIOTIC OINTMENT**

Each gram contains:
'Aerosporin'® brand Polymyxin B Sulfate 5,000 Units
Neomycin Sulfate ..... 5 mg.
Zinc Bacitracin ..... 400 Units
in a special petrolatum base.



'POLYSPORIN'®
brand **ANTIBIOTIC OINTMENT**

Offers combined antibiotic action for treating conditions due to susceptible organisms amenable to local medication.

Each gram contains:
'Aerosporin'® brand
Polymyxin B Sulfate ..... 10,000 Units
Zinc Bacitracin ..... 500 Units
in a special petrolatum base.

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N. Y.

Squibb Announces Chemipen

Squibb Alpha-Phenoxyethyl Penicillin Potassium

new chemically improved penicillin which provides the highest blood levels that are obtainable with oral penicillin therapy



As a pioneer and leader in penicillin therapy for more than a decade, Squibb is pleased to make Chemipen, a new chemically improved oral penicillin, available for clinical use.

With Chemipen it becomes possible as well as convenient for the physician to achieve and maintain higher blood levels—with greater speed—than those produced with comparable therapeutic doses of potassium penicillin V. In fact, Chemipen is shown to have a 2:1 superiority in producing peak blood levels over potassium penicillin V.*

Extreme solubility may contribute to the higher blood levels that are so notable with Chemipen.* Equally notable is the remarkable resistance to acid decomposition (Chemipen is stable at 37°C. at pH 2 to pH 3), which in turn makes possible the convenience of oral treatment.

And the economy for your patients will be of particular interest—Chemipen costs no more than comparable penicillin V preparations.

Dosage: Doses of 125 mg. (200,000 u.) or 250 mg. (400,000 u.), t.i.d., depending on the severity of the infection. The usual precautions must be carefully observed with Chemipen, as with all penicillins. Detailed information is available on request from the Professional Service Department.

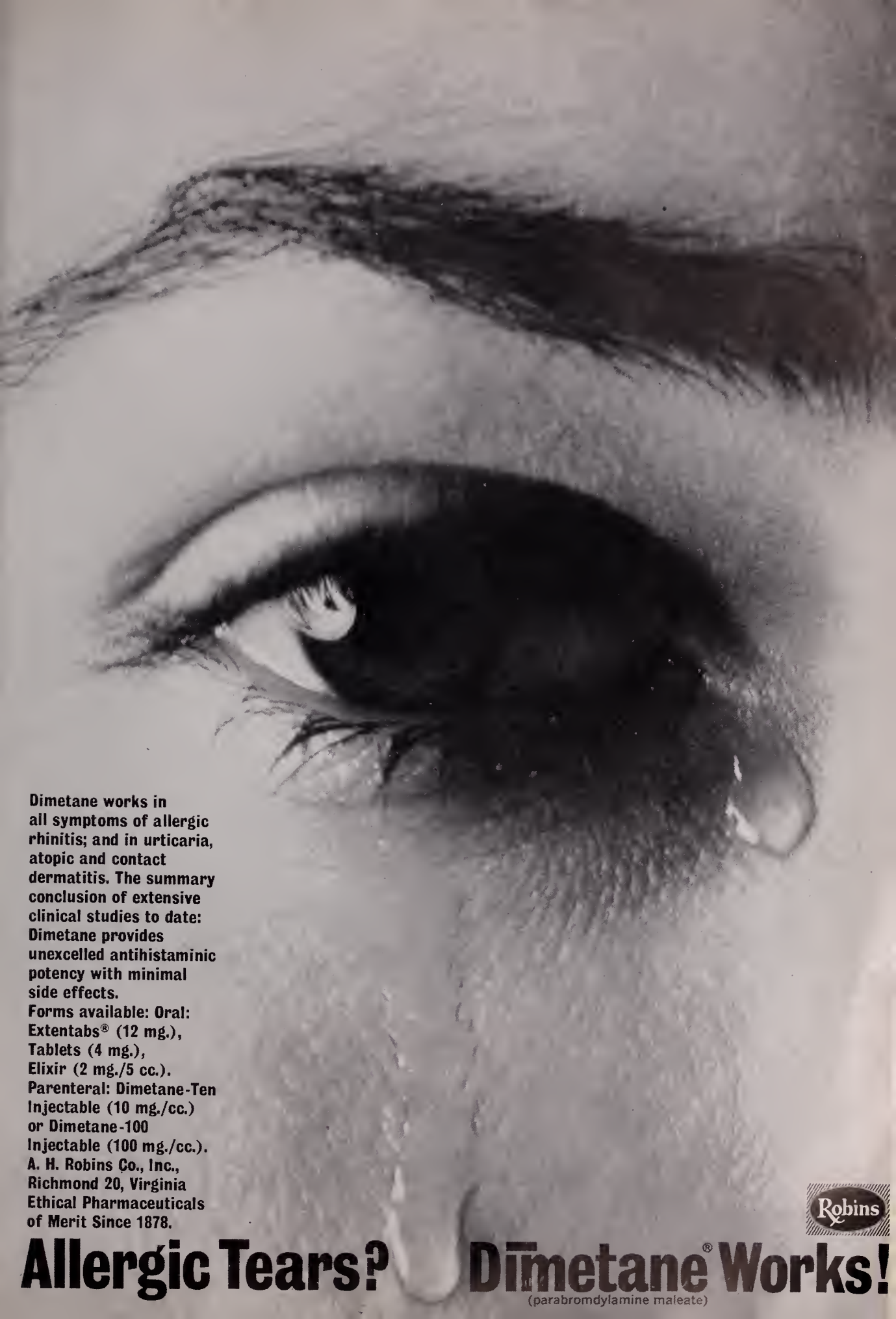
Supply: Chemipen Tablets of 125 mg. (200,000 u.) and 250 mg. (400,000 u.), bottles of 24 tablets. Chemipen Syrup (cherry-mint flavored, nonalcoholic), 125 mg. per 5 cc., 60 cc. bottles.

*Knudsen, E. T., and Rolinson, G. N.: Lancet 2:1105 (Dec. 19) 1959.

"CHEMIPEN" IS A SQUIBB TRADEMARK.

SQUIBB

Squibb Quality—the Priceless Ingredient



Dimetane works in all symptoms of allergic rhinitis; and in urticaria, atopic and contact dermatitis. The summary conclusion of extensive clinical studies to date: Dimetane provides unexcelled antihistaminic potency with minimal side effects.
Forms available: Oral: Extentabs® (12 mg.), Tablets (4 mg.), Elixir (2 mg./5 cc.). Parenteral: Dimetane-Ten Injectable (10 mg./cc.) or Dimetane-100 Injectable (100 mg./cc.).
A. H. Robins Co., Inc., Richmond 20, Virginia
Ethical Pharmaceuticals of Merit Since 1878.

Robins

Allergic Tears? Dimetane® Works!

(parabromdylamine maleate)



Beating too fast?

Slow it down with SERPASIL®

(reserpine CIBA)

Serpasil has proved effective as a heart-slowing agent in the following conditions: mitral disease; myocardial infarction; cardiac arrhythmias; neurocirculatory asthenia; thyroid toxicosis; excitement and effort syndromes; cardiac neurosis; congestive failure. Serpasil should be used with caution in patients receiving digitalis and quinidine. It is not indicated in cases of aortic insufficiency.

SUPPLIED: Tablets, 0.1 mg., 0.25 mg. (scored) and 1 mg. (scored). Complete information available on request.

CIBA

OPEN UP THE SINUSES

By Deep Decongestion

Neo-Vadrin is the deep nasal decongestant that restores drainage and ventilation of all eight sinuses by opening up the obstructed ostia.

The therapeutic effect is both prompt and prolonged. Hence Neo-Vadrin is ideal for treatment of deep nasal and sinus congestion due to head colds, hay fever, vasomotor rhinitis, nasal allergy, and sinusitis.

In addition, Neo-Vadrin is antiseptic and locally anesthetic. May be used by patients of all ages.

dl-Norephedrine HCl	0.4%
Phenylephrine HCl	0.15%
Chlorobutanol	0.15%
Benzalkonium Chloride	0.005%

The more prolonged action of dl-norephedrine[1] combined with prompt effective phenylephrine[2] provides a long span of hours for more satisfactory therapeutic response.

Supplied in 1 fld. oz. dropper bottles with special nasal applicator and ¾ oz. plastic spray bottles.

NEO-VADRIN

FOR NASOSINUSITIS

Samples are available to physicians upon request. Please make your request on your prescription sheet or letterhead.

1. **Sollmann's Manual of Pharmacology,** 8th ed., 1957, p. 507.
2. **Modern Drug Encyclopedia,** 7th ed., 1958, p. 757.

FIRST TEXAS *Pharmaceuticals, Inc.*

DALLAS **Since 1901** **ATLANTA**

IN CONTRACEPTION...



PHOTOGRAPH: HUMAN SPERMATOZOA 400X. HEMOCYTOMETER DIFFUSION TEST.

WHY IS DIFFUSION IMPORTANT?

Because the active ingredients of a spermicidal preparation must diffuse rapidly into the seminal clot and throughout the vaginal canal to be clinically effective. Lanesta Gel offers this *dual* protection. Its four spermicidal agents quickly invade the clot to stop the main body of sperm. It spreads evenly and quickly throughout the vaginal canal—seeks out every wrinkle and fold that may offer concealment to sperm. With this rapid diffusion, your patient receives full benefit of the swift spermicidal action of Lanesta Gel — in minutes — a decisive measure in conception control.

In Lanesta Gel *7-chloro-4-indanol,* a new, effective, nonirritating, nonallergenic spermicide, produces immediate immobilization of spermatozoa in dilution of up to 1:4,000. The addition of 10 per cent NaCl in ionic form greatly accelerates spermicidal action. Ricinoleic acid facilitates rapid inactivation and immobilization of spermatozoa and sodium lauryl sulfate acts as a dispersing agent and spermicidal detergent.

Lanesta Gel with a diaphragm provides one of the most effective means of conception control. However, whether used with or without a diaphragm, the patient and you, doctor, can be certain that Lanesta Gel provides faster spermicidal action — plus essential diffusion and retention of the spermicidal agents in a position where they can act upon the spermatozoa.



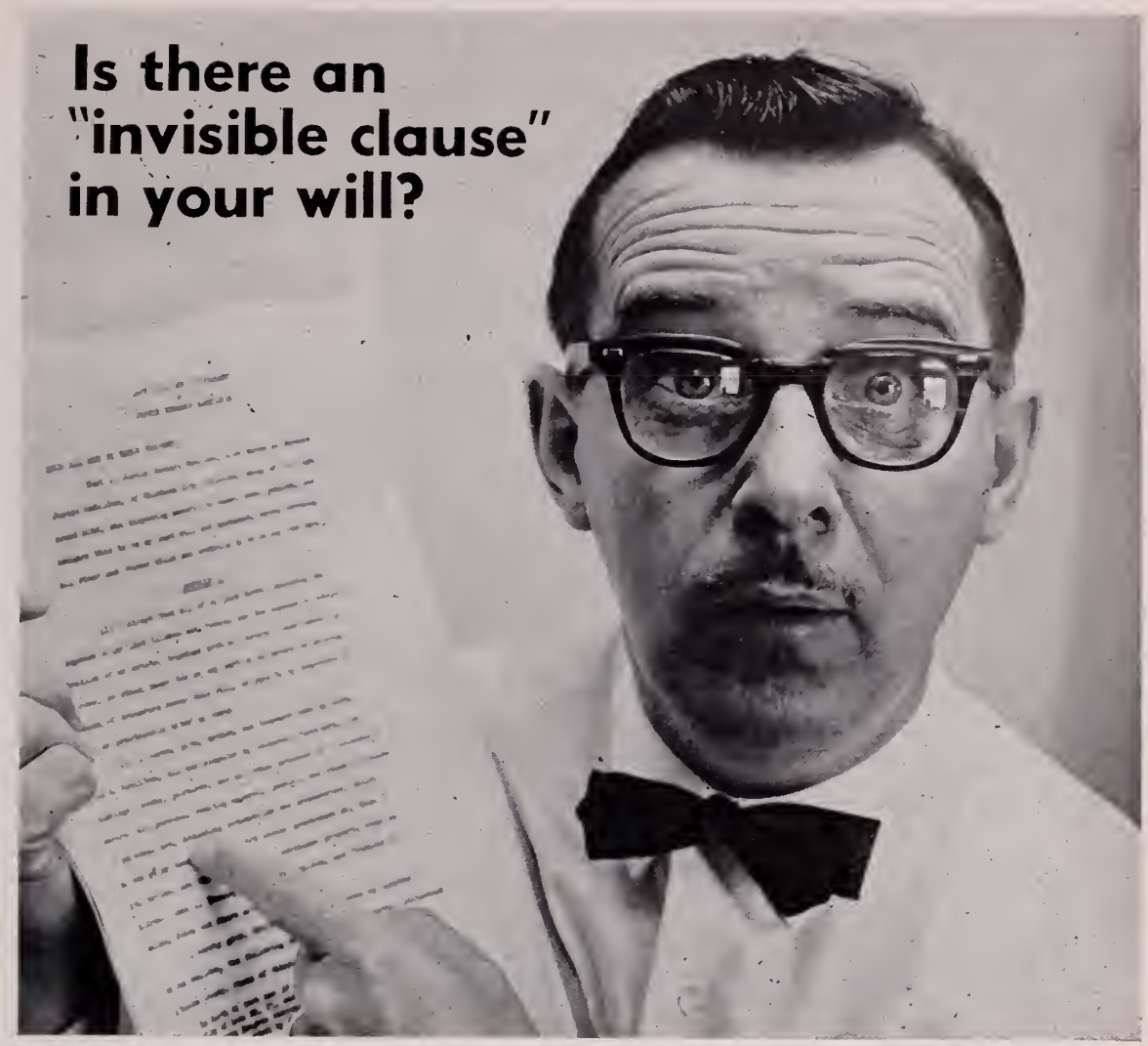
new Lanesta® Gel

Supplied: Lanesta Exquiset® . . . with diaphragm of prescribed size and type; universal introducer; Lanesta Gel, 3 oz. tube, with easy clean applicator, in an attractive purse. Lanesta Gel, 3 oz. tube with applicator; 3 oz. refill tube — available at all pharmacies.

A product of Lanteen® research.

Manufactured by Esta Medical Laboratories, Inc., Alliance, Ohio. Distributed by GEORGE A. BREON & CO., New York 18, N. Y

Is there an "invisible clause" in your will?



"I hereby accidentally bequeath $20,000 of my estate to the tax collector."

"I hereby give the difficult task of managing substantial sums of money to those members of my family who are absolutely unqualified for this task."

You won't find those clauses written in any will. But they may be there anyway. You can guard against this problem with a trust under your will. Let us explain the family security and tax saving advantages of a trust under your will. A conference costs you nothing, yet it may save your estate many thousands of dollars.
Send for Liberty's free booklet "A Trust Under Your Will — for family security and tax saving." There's no obligation.

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Proven

in over five years of clinical use and more than 750 published clinical studies

Effective

for relief of anxiety and tension

Outstandingly Safe

- simple dosage schedule produces rapid, reliable tranquilization without unpredictable excitation
- no cumulative effects, thus no need for difficult dosage readjustments
- does not produce ataxia, change in appetite or libido
- does not produce depression, Parkinson-like symptoms, jaundice or agranulocytosis
- does not impair mental efficiency or normal behavior



when that early Monday morning telephone call is from a weekend do-it-yourselfer

"...and this morning, Doctor, my back is so stiff and sore I can hardly move."

now...there is a way to prompt, dependable relief of back distress

the pain goes while *the muscle relaxes*

POTENT — rapid relief in acute conditions

SAFE — for prolonged use in chronic conditions

notable safety — extremely low toxicity; no known contraindications; side effects are rare; drowsiness may occur, usually at higher dosages

rapid action, sustained effect — starts to act quickly, relief lasts up to 6 hours

easy to use — usual adult dosage is one 350 mg. tablet 3 times daily and at bedtime

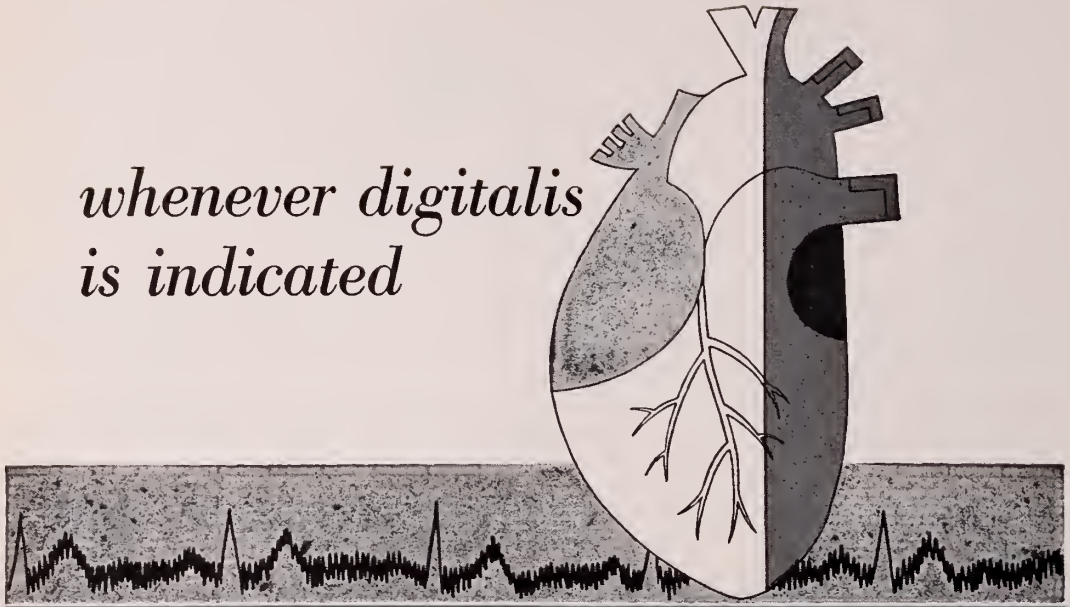
supplied — as 350 mg., white, coated tablets, bottles of 50; *also available for pediatric use:* 250 mg., orange capsules, bottles of 50

® WALLACE LABORATORIES, New Brunswick, New Jersey

SOMA™

(CARISOPRODOL WALLACE)

whenever digitalis is indicated



'LANOXIN'® brand DIGOXIN

formerly known as Digoxin 'B. W. & Co.'®

"If one digitalis agent were to be recommended for its adaptability to the many and varied clinical contingencies, we believe Digoxin would be the drug of choice."

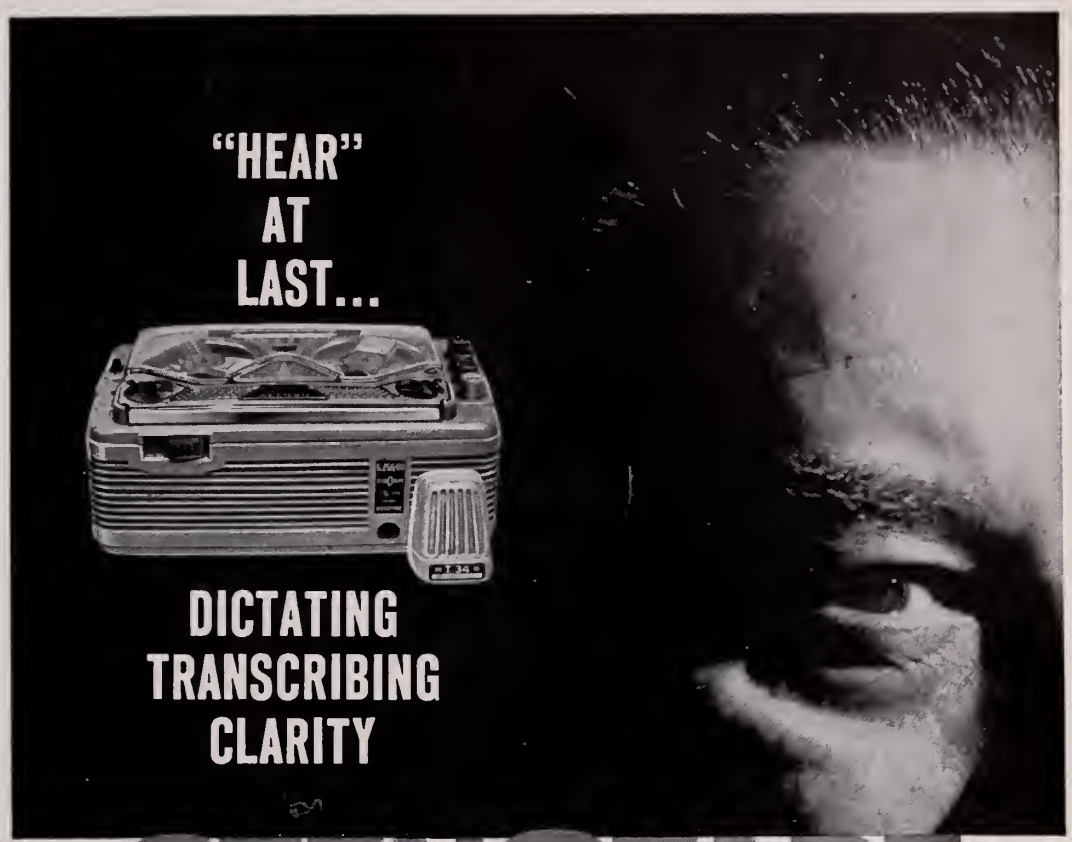
Lown, B., and Levine, S. A.: Current Concepts in Digitalis Therapy, Boston, Little, Brown & Company, 1954, p. 23, par. 2.

'LANOXIN' TABLETS
0.25 mg. scored (white)
0.5 mg. scored (green)

'LANOXIN' INJECTION
0.5 mg. in 2 cc. (I.M. or I.V.)

'LANOXIN' ELIXIR PEDIATRIC
0.05 mg. in 1 cc.

BURROUGHS WELLCOME & CO. (U.S.A.) INC., Tuckahoe, N.Y.



STENOTAPE GELOSO

offers true "professional" dictating transcribing sound and efficiency

Doctor, Lawyer, Office Chief...here is the soundest practice you can establish to end paper-work problems. LISTEN: StenOtape gives you the greatest clarity of sound in the dictating field today. This 6½ lb. compact unit, with its extremely sensitive microphone records every word perfectly within a 30 foot radius. You can actually dictate comfortably from any point in the room. Seated and relaxed, you can tape interviews with a patient or client; and because of StenOtape's unique sound-fidelity, your secretary will hear and enjoy every word of your error-free dictation. Doctors and Dentists can play their post graduate educational tapes on StenOtape and enjoy superb playback quality. At the office, home or away, StenOtape records everything up to 2 hours on one tape...phone calls, conferences, dictation, *even music!* Hear the StenOtape difference now...it's an exceptional value!

only $179.95

FULL YEAR GUARANTEE Federal Tax Included

Check These Other Major StenOtape Features:

● Accurate word-counter. ● Built-in Speaker. ● 4" high, weighs only 6½ lbs. ● Travels in handsome attache case. ● Low-cost accessories available to cover every dictating-transcribing-recording situation. ● Precision designed by Geloso, Europe's largest integrated electronics manufacturer of communication equipment. ● Sales and Service Coast to Coast.

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251 Park Ave. So., Dept. 85, New York 10, N. Y

Gentlemen: Please rush, without obligation, illustrated booklet "The Facts About Dictating Machines." I understand that should I decide to purchase a StenOtape this coupon entitles me to a lifetime supply (6 rolls) of reusable Magnetic Tape worth $15.00.* **Offer expires July 31, 1960*

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to relieve itching, burning skin lesions
just press the button on the can

Schering

METI-DERM® AEROSOL

prednisolone topical

for all steroid-responsive skin lesions · available with or without neomycin

5-472

there's
a better
move
than
scratching...





no irritating crystals[1] · uniform concentration in each drop[2]

STERILE OPHTHALMIC SOLUTION

NEO-HYDELTRASOL®

PREDNISOLONE 21-PHOSPHATE-NEOMYCIN SULFATE

2,000 TIMES MORE SOLUBLE THAN PREDNISOLONE OR HYDROCORTISONE

"The solution of prednisolone has the advantage over the suspension in that no crystalline residue is left in the patient's cul-de-sac or in his lashes . . . . The other advantage is that the patient does not have to shake the drops and is therefore sure of receiving a consistent dosage in each drop."[2]

1. Lippmann, O.: Arch. Ophth. **57**:339, March 1957.
2. Gordon, D.M.: Am. J. Ophth. **46**:740, November 1958.

supplied: 0.5% Sterile Ophthalmic Solution NEO-HYDELTRASOL (with neomycin sulfate) and 0.5% Sterile Ophthalmic Solution HYDELTRASOL*. In 5 cc. and 2.5 cc dropper vials. Also available as 0.25% Ophthalmic Ointment NEO-HYDELTRASOL (with neomycin sulfate) and 0.25% Ophthalmic Ointment HYDELTRASOL. In 3.5 Gm. tubes.

HYDELTRASOL and NEO-HYDELTRASOL are trademarks of Merck & Co., Inc.

MSD MERCK SHARP & DOHME Division of Merck & Co., Inc., Philadelphia 1, Pa.

sulfa therapy suited to young tastes and tempers...



Employs the N^1 acetyl form of KYNEX to impart high palatability yet retain single-daily-dose effectiveness and rapid, high sustained action against sulfa-susceptible infections. **Dosage:** first day, 1 tsp. (250 mg) for each 20 lbs.; thereafter, ½ tsp. daily for each 20 lbs. For 80 lbs., use adult dosage of 4 tsp. (1.0 Gm.) initially; and 2 tsp. (0.5 Gm.) thereafter. Taken once a day—preferably after a meal. **Supplied:** Each tsp. (5 cc.) contains 250 mg. sulfamethoxypyridazine activity. Bottles of 4 and 16 fl. oz.

CHERRY LIQUID AND 1-DOSE-DAILY

KYNEX®

N^1 Acetyl Sulfamethoxypyridazine

ACETYL PEDIATRIC SUSPENSION

Lederle LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York



Diagnostic Quandaries

Colitis? Gall Bladder Disease?
Chronic Appendicitis?
Rheumatoid Arthritis? Regional Enteritis?

A DISEASE that is frequently overlooked in solving diagnostic quandaries is amebiasis. Its symptoms are varied and contradictory, and diagnosis is extremely difficult. In one study, 56% of the cases would have been overlooked if the routine three stool specimens had been relied on.[1]

Another study found 96% of a group of 150 patients with rheumatoid arthritis were infected by *E. histolytica*. In 15 of these subjects, nine stool specimens were required to establish the diagnosis.[2]

Webster discovered amebic infection in 147 cases with prior diagnoses of spastic colon, psychoneurosis, gall bladder disease, nervous indigestion, chronic appendicitis, and other diseases. Duration of symptoms varied from one week to over 30 years. In some cases, it took as many as six stool specimens to establish the diagnosis of amebiasis.[3]

Now treatment with Glarubin provides a means of differential diagnosis in suspected cases of amebiasis. Glarubin, a crystalline glycoside obtained from the fruit of *Simarouba glauca*, is a safe, effective amebicide. It contains no arsenic, bismuth, or iodine. Its virtual freedom from toxicity makes it practical to treat suspected cases without undertaking difficult, and frequently undependable, stool analyses. Marked improvement following administration of Glarubin indicates pathologically significant amebic infection.

Glarubin is administered orally in tablet form and does not require strict medical supervision or hospitalization. Extensive clinical trials prove it highly effective in intestinal amebiasis.

Glarubin*

TABLETS

specific for intestinal amebiasis

Supplied in bottles of 40 tablets, each tablet containing 50 mg. of glaucarubin.

Write for descriptive literature, bibliography, and dosage schedules.

1. Cook, J.E., Briggs, G.W., and Hindley, F.W.: Chronic Amebiasis and the Need for a Diagnostic Profile, Am. Pract. and Dig. of Treat. *6*:1821 (Dec., 1955).

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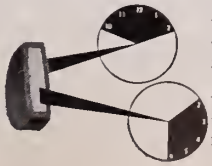
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References: 1. Fabricant, N. D.: E. E. N. T. Monthly **37**:460 (July) 1958. 2. Lhotka, F. M.: Illinois M. J. 112:259 (Dec.) 1957. 3. Farmer, D. F.: Clin. Med. 5:1183 (Sept.) 1958. 4. Fuchs, M.; Bodi, T.; Mallen, S. R.; Hernando, L., and Moyer, J. H.: Antibiotic Med. & Clin. Ther. 7:37 (Jan.) 1960. 5. Halpern, S. R., and Rabinowitz, H.: Ann. Allergy 18.36 (Jan.) 1960.

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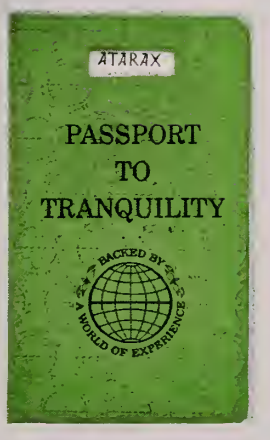
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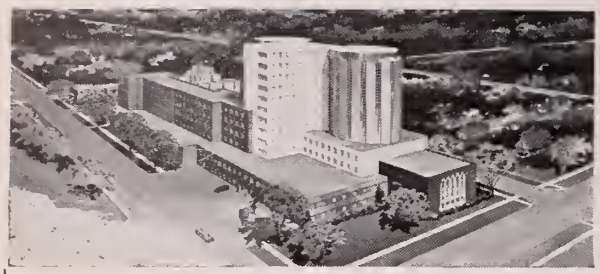
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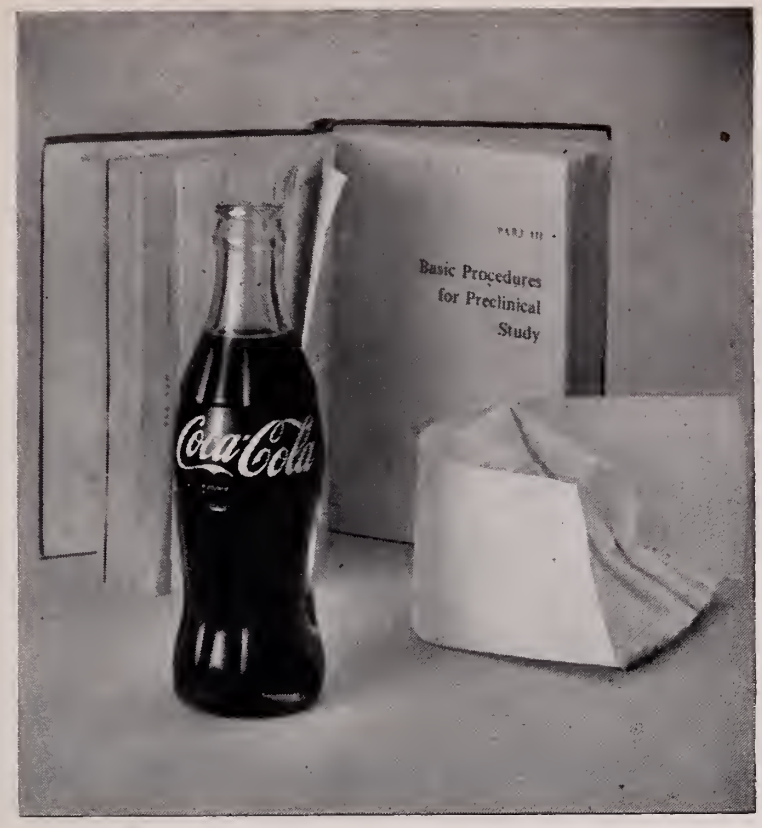
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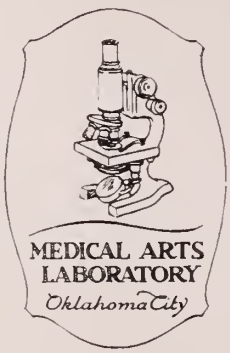
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Index to Advertisers

Ames Company, Inc. ... inside back
American Geloso Electronics, Inc. ... xxxv
Arlington Hotel ... xlix

Beverly Hills Clinic ... lix
Bone and Joint Hospital ... liii
George A. Breon & Co. ... xxx
Bristol Laboratories, Inc. xviii and xix, xxii and xxiii
Burroughs-Wellcome & Company, Inc. ... xxv, xxxiv

Ciba ... xxviii
Coca Cola Bottlers ... xlix
Coyne Campbell Sanitarium ... liii
Credit Service ... xlix

First Texas Pharmaceuticals, Inc. ... xxix

Geigy ... xi
Goldfain Laboratory ... liv

J. E. Hanger ... xlvii

Drs. Lain, Lamb and Jones ... liv
Lederle Laboratories ... 460 and 461, xxi, xxxviii, xlii, xliii, xlvii, xlviii, l, lix
Liberty National Bank & Trust Co. ... xxxi
Eli Lilly & Company ... xvi

Medical Arts Laboratory ... lii
S. E. Massingill Company ... xxxix
Melton Co., Inc. ... l
Merck Sharp and Dohme, Inc. ... iv, xxxvii
McAlester Clinic ... lv
Midwest Surgical Supply Co., Inc. ... lviii

Neurological Hospital ... lix
North American Company ... xl

Oklahoma Allergy Clinic ... lv
Osler Radioisotope Laboratory ... lviii

Parke Davis & Company ... inside front and iii
Pharmaceutical Manufacturers Association 464 and 465
Physicians Casualty & Health Assn. ... xlviii
Physicians Laboratory ... lviii
Pitman-Moore Company ... xx
A. H. Robbins Co., Inc. ... viii and ix, xxvii, xli
Rock Haven Hospital ... lix
Roerig ... vi, xlvi
St. Joseph Hospital ... xlviii
St. Paul Companies ... xlvii
Sardeau, Inc. ... xiv
W. B. Saunders Company ... vii
G. D. Searle & Company ... 445
Schering Corporation ... xv, xxxvi, xliv and xlv, li
Seven-Up Bottlers ... l
Smith-Dorsey ... xxiv, xliii
Smith, Kline & French Laboratories ... outside back
Springer Clinic ... lvi
E. R. Squibb and Sons ... xxvi
Stout Clinical Laboratory ... lvi
Sugg Clinic ... lvii
Terrell's Laboratories ... lviii
Tulsa Clinic ... lvii
S. J. Tutag & Co. ... li
Wallace Laboratories ... x, xxxii, insert, xxxiii
Wesson Oil ... xii and xiii
Winthrop Laboratories ... xvii, insert

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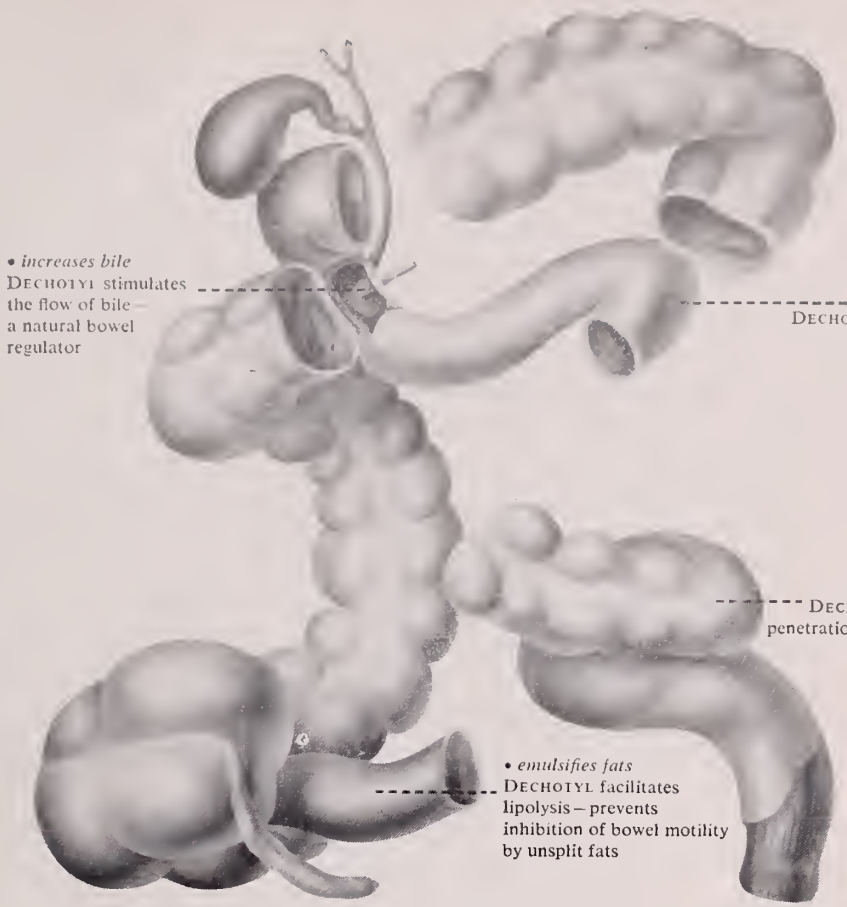
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